

THE NEURAL SUBSTRATE OF EMOTION IN MAN: A study in methodology

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Emotional behaviour has several components, which include emotional perception, emotional expression, autonomic reactivity, and inner subjective experience. It is hypothesised that these different processes can be selectively disturbed after brain damage. However, emotional and cognitive deficits usually co-exist in a particular patient, and the relationship between cognition and emotion is discussed.

Animal data indicate that, following an initial sensory analysis in primary and association cortex, a stimulus acquires emotional significance by interfacing with "limbic" processes through multimodal cortex. Emotional expressions also develop in the course of similar multistage integrations between motor and limbic processes. This suggests three possible relationships between cognitive and emotional deficit. First, an inability to either perceive or express emotion may be directly due to various primary "non-emotional" perceptuo-motor deficits. Secondly, damage to neural sites where limbic and sensori-motor systems interface may produce deficits which are simultaneously cognitive and emotional. For example, perseverative behaviour in the Wisconsin Card Sorting Test and inability to control emotional impulses may be different aspects of a single deficit, which is indissociably cognitive and emotional. Thirdly, there may be a class of "pure" emotional deficits, which are not associated with perceptuo-motor or cognitive deficit.

Tests based on the above classification approach were used to assess the effect of lesion site on emotional functioning. Cognitive function, emotional perception, emotional expression, subjective response, and autonomic reactivity were assessed in 48 patients with focal brain lesions, and 10 matched non-brain injured controls. Patients with anterior lesions were impaired relative to posteriors and controls in emotional perception and expression. These frontal deficits remained after statistical control of lesion variables (size, aetiology, and degree of bilateral involvement), and non-emotional perceptual, motor or cognitive impairments. Though such statistical controls provide suggestive evidence of a specific emotional deficit, they cannot completely eliminate the possibility that the frontal deficit is secondary to perceptuo-motor or cognitive impairment. Experimental strategies designed to provide more positive evidence of a specific emotional disorder were employed, and met with partial success.

Dysphasia affected performance on tests of emotional perception and expression after left brain damage. Following right hemisphere damage, visual perceptual deficit contributed to impairment in emotional perception. There were indications that hemispheric perceptual-cognitive asymmetries may not account for all right-left differences on the emotional subtests. However no current model of hemispheric emotional asymmetries could fully explain the present findings. For example, the data on emotional perception supported the view that the right hemisphere is specialised for attentional/emotional processes (Heilman et al, 1983), but neglect and emotional expression were not correlated. Also, the present data generally contradicted the hypothesis that positive and negative emotions are lateralised in the left and right hemispheres, respectively.

The final chapter assesses the extent to which the data fit the proposed system for categorising emotional disorder. Limitations to the methods used in making this evaluation were discussed, and possible ways of overcoming these restrictions were briefly considered.



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### 1.1. The Problem

Emotional disorders are common in patients after brain damage of diverse aetiology (Lishman, 1978; Benson and Blumer, 1975), including stroke (Magni and Schifano, 1984; Robinson and Price, 1982), head injury (Levin *et al.*, 1982; Weddell *et al.*, 1980), epilepsy (Hermann and Whitman 1984; Bear and Fedio, 1977), cerebral tumour (Whitlock, 1978; Hecaen, 1964), and degenerative neurological diseases such as parkinsonism and multiple sclerosis (Mindham, 1974; Surridge, 1969).

The emotional disturbance displayed in the particular patient is determined by many factors. For our purposes we can categorise these factors under the two broad headings of "lesion variables" and "psychosocial influences". Lesion site is the variable which has so far received most attention. Characteristic emotional syndromes have been reported to be particularly associated with frontal lobe damage (Lishman, 1968; Hecaen, 1964), temporal lobe damage (Taylor and Falconer, 1968), and damage to right and left hemispheres (Tucker, 1981).

The second general class of influences which determine the emotional disturbances in the brain damaged patient are loosely categorised as psychosocial, or alternatively psychiatric-reactive. Patient's emotional reactions are determined in part by the precise characteristics of their neurological deficit. For example, the word finding difficulties of the dysphasic patient can be extremely frustrating (Benson, 1973). In addition, emotional reactions also

depend on premorbid emotional adjustment (Lishman, 1973), which is a function of genetic factors (Slater and Roth, 1969), early personal history (Freud, 1971), current family relationships (Paolino and McCrady, 1979; Vaughn and Leff, 1976), and wider social influences (Brown and Harris, 1978).

In the individual brain damaged patient it is possible that some emotional symptoms are directly related to lesion factors, and have less close causal links with psychosocial influences. Conversely, other emotional symptoms may be more directly determined by psychosocial factors, with lesion variables exerting a relatively minor influence. For some emotional disorders, both groups of factors may combine and interact in a complex (and as yet poorly understood) fashion. For example, we know that psychosocial factors are important determinants of depression (Campbell *et al.*, 1983; Paykel *et al.*, 1980), and it has been suggested that depression is particularly common after left anterior damage (Robinson and Szetela, 1981). If lesion and psychosocial factors interact, we might find that the combination of anterior lesion site and adverse social circumstances leads to particularly severe depression.

While we may be able to draw theoretical distinctions between psychosocial and lesion variables, our knowledge about the specific effects of the many possible psychosocial and lesion variables is currently rudimentary. Given this circumstance, we need to greatly simplify the problem. Therefore in this present investigation of the neural substrate of emotion, the contribution of psychosocial influences to emotional symptoms are minimised by the experimental design employed. To investigate the emotional consequences of focal brain damage, a system for classifying emotional deficit is developed, and methods for investigating this system are proposed. The performance on emotional tests of patients with anterior cerebral lesion is contrasted with the emotional test scores of patients who have posterior cerebral damage. We also

ask whether the right and left hemispheres have different roles in the regulation of emotional processes.

## **1.2. Intra-hemispheric Lesion Site and Emotional Deficit**

### **Frontal lobes and social-emotional behaviour**

Tow (1955) extensively reviewed early investigations which indicated that damage to the frontal lobes was particularly liable to produce a range of emotional and behavioural symptoms, including alterations in mood, euphoria, depression, tactlessness, and "moral defects". Similar behavioural emotional changes were also observed subsequent to prefrontal leucotomy, a neurosurgical procedure in vogue in the middle of the present century for the treatment of psychiatric disorders. Partridge (1950) reported that after the association fibres between the prefrontal cortex and other cerebral structures are bilaterally sectioned patients were sometimes euphoric; complained of a general loss of feeling; they lacked initiative and spontaneity; and lost restraint – sometimes becoming "greedy" or more outspoken. Frontal tumour (Hecaen, 1964; Rylander, 1939) and subarachnoid haemorrhage from an anterior communicating artery aneurysm (Storey, 1970; Logue *et al.*, 1969) have also been reported to cause quite similar emotional disturbances. In fact, a number of studies carried out throughout this century on patients with lesions of varying aetiology have indicated that a distinct syndrome of emotional and behavioural symptoms are particularly evident following frontal lobe damage. On the one hand, this consistency over a long period of study encourages us to suppose that certain emotional disorders are particularly linked with frontal lobe damage. On the other hand the remarkable similarity of earlier observations with present day descriptions suggest that our progress in

defining the nature of the frontal lobe syndrome has been somewhat limited.

Our lack of progress is probably, in part, due to the difficulties in isolating, defining and quantifying the abnormal emotional behaviours associated with frontal lobe damage. However ethologists have developed reliable quantitative measures of social and emotional behaviours in primates, and in relatively recent years these objective ethological measures have been applied in the study of the social behaviour of monkeys with focal cerebral lesions. Franzen and Myers (1973) studied the social behaviour of rhesus monkeys with bilateral removal of the prefrontal cortex. They observed the animals in quasi-natural social colonies, and pre-operative and post-operative social behaviour were compared. Their prefrontal animals showed profound social disturbances. Post-operatively facial expression and vocalisations were generally markedly reduced. They avoided social contact and this was taken to indicate that the prefrontal monkeys were more fearful. Maternal behaviour was disrupted. For example, the infant might be allowed the nipple, but it was not held. On some occasions the infant was stepped on while still holding the nipple as the mother initiated a bout of hyperactivity. Deets *et al.* (1970) characterised some behaviours as being "inappropriate". Together with Franzen and Myers they noted that some animals would display aggression without any apparent provoking stimulus from other monkeys, also when the dominant monkey made a sexual approach the prefrontal female behaved unpredictably and either threatened him, or ignored him, or ran away.

Franzen and Myers (1973) and Deets *et al.* (1970) report on animals with large prefrontal ablations. However some studies have considered the consequences of more restricted bilateral lesions of the orbital-frontal cortex. Butter and Snyder (1972) report on a series of experiments investigating rhesus monkeys with orbital frontal lesions. They concluded that in some, but not all,



situations removal of the orbital frontal cortex enhanced aversive reactions to threatening stimuli. This lesion also reduced aggressive reactions in several threatening situations, and when the animals were placed in a social colony they quickly fell to the bottom of the dominance hierarchy. This reduction in aggression contrasted with the unaltered aggressive behaviours of the sham operated control monkeys or the superior temporal and inferotemporal lesioned groups. Kamback (1973) made similar observations, while Kling and Steklis (1976) report that in addition to reduced aggression their orbital group also demonstrated a whole range of deficient social behaviours. Raleigh *et al.* (1981 and 1979) report on social and emotional changes in vervet monkeys with orbital frontal lesions. Significant alterations in aggressive behaviour were observed, but they did not find the decrease in frequency of aggressive encounters that have been noted in rhesus monkeys after orbital-frontal ablation.

The social consequences of selective dorsolateral frontal ablation have also been studied. While Butter and Snyder (1972) reported no changes in emotional behaviour following dorsolateral frontal lesions, Kamback (1973) found an increased aggression in the pigtail monkey. Kling and Steklis (1976) also found increased aggression, in addition they reported a lack of facial expression and a reduction in social contact. Kling and Steklis described a female rhesus monkey with bilateral dorsolateral frontal ablations who rejected and abandoned her infant and subsequently left the social group. In contrast Miller (1976) did not observe a generalised disturbance of social behaviours. His dorsolateral frontal rhesus monkeys continued to maintain similar levels of affiliative responses such as grooming and time together. However Miller (1976) did agree with Kling and Steklis (1976) that there was increased aggression and a lack of facial expression.

The social consequences of damage to the anterior cingulate gyrus have also been given some attention. Cingulectomy has been advocated for the treatment of psychiatric illness (Ballantine *et al.*, 1977); in some primate studies lesions of the anterior cingulate gyrus produced profound changes in the social behaviour of monkeys (Glees *et al.*, 1950); and the anterior cingulate gyrus appears to have a role in the voluntary initiation of affective vocalisations in the primate (Kirzinger and Jurgens, 1982; Jurgens, 1979). However Franzen and Myers, (1973) and Mirsky *et al.* (1957) found no social or emotional changes after cingulectomy, and it is concluded that the social consequences of cingulectomy are, at present, equivocal.

In sum, social-emotional deficits have been consistently observed in man and monkey subsequent to prefrontal damage. There have been various attempts to distinguish the emotional sequelae of more restricted anterior lesions. Though further work with animal and human lesion subjects is required, it is possible that dorsolateral and orbito-frontal ablation produce different emotional symptoms. The role of the anterior cingulate gyrus in the regulation of emotional behaviour also remains to be defined. Furthermore, in spite of the consistent findings of emotional deficit after prefrontal damage different studies do not always agree about the precise nature of the emotional sequelae. One reason for this is that different primate species have been studied. Rhesus, pigtail, and vervet monkeys all display species-specific social behaviours, and so the effect of prefrontal damage on social behaviour will depend on the monkey species. Differences between species are likely to be greatest when comparing monkey and man.

## Temporal lobes and social-emotional behaviour

The temporal lobes have also been implicated in the regulation of emotional behaviour. Kluver and Bucy (1939) identified several sequelae of bilateral temporal lobectomy in rhesus monkeys. These included a decrease in belligerence, a reduction of fear toward normally fear-inducing objects, and hypersexuality. Though the full Kluver-Bucy syndrome is rarely observed in man (Marlowe *et al.*, 1975), human patients sometimes exhibit features of the syndrome after bilateral temporal damage (Poeck, 1967).

These social and emotional disturbances appear to be particularly associated with damage to anterior temporal cortex (Raleigh *et al.*, 1981; Kling and Steklis, 1976; Franzen and Myers, 1973). Franzen and Myers (1973) studied the social behaviour of rhesus monkeys with bilateral removal of anterior temporal cortex, sparing the amygdalae. Post-operatively lesioned monkeys became socially isolated; were less aggressive; with maternal and other social behaviours seriously disrupted. These workers also found that their monkeys with anterior temporal lobectomies showed marked reductions in facial expressions and spontaneous vocalisations.

Similar emotional disorders are found after amygdalectomy. Rhesus monkeys with bilateral amygdalectomy become less aggressive, but are less fearful of man (Kling, 1972). These animals avoid social contact and it has been suggested that this is because they are more fearful of other monkeys. The reduced aggression and possible increased fearfulness perhaps accounts for the tendency of animals with anterior temporal lobectomy and/or amygdalectomy to fall to the bottom of the dominance hierarchy (Myers, 1972; Kling, 1972; Plotnik, 1968; Rosvold *et al.*, 1954). Mark *et al.* (1972) describe an epileptic patient who made violent stabbing attacks in association with

abnormal EEG activity in both amygdalae. Further, electrical stimulation of the right amygdala produced the aggressive attack behaviour seen in the epileptic ictus, and a small lesion in both amygdalae reduced the frequency of rage attacks and epileptic seizures. Narabayashi *et al.* (1963) and Vaernet and Madsen (1970) also found that stereotactic amygdalotomy could reduce aggressive behaviour in patients with severe behavioural disorder. Further, aggressive behavioural disorders are less common after anterior temporal lobectomy for the relief of epilepsy (Jenson and Larsen, 1979; Falconer, 1973). Of course, the human temporal lobe is not only involved in the regulation of aggressive behaviour. On electrical stimulation of medial temporal structures, Penfield (1955) and Halgren *et al.* (1978) report other emotional experiences in human patients, such as anxiety and fear.

In man, evidence for a link between the temporal lobes and emotional behaviour has also been sought in the study of the psychiatric symptoms of patients with temporal lobe epilepsy. Slater and Beard (1963) concluded that temporal lobe epilepsy could produce schizophrenic psychosis. A high incidence of neurotic and behavioural disturbance has also been noted in many investigations of temporal lobe epilepsy (Shukla *et al.*, 1979; Jenson and Larsen, 1979; Taylor and Falconer, 1968). On the other hand, we must remember that psychiatric symptoms in the brain damaged are determined by a range of psychosocial factors in addition to lesion variables. The difficulty of effectively controlling these confounding psychosocial factors, is a serious obstacle in the investigation of the relationship between lesion site and psychiatric symptoms (Hermann and Whitman, 1984; Kligman and Goldberg, 1975). Also, there are considerable difficulties in getting a representative sample of temporal lobe epileptics. However Small *et al.* (1966) and Stevens (1966) both compared a group of patients who had temporal lobe epilepsy with a group, matched in

terms of psychosocial variables, who had non-temporal epilepsy. They found a high incidence of psychopathology in both epileptic groups, and concluded that the psychiatric disorder is largely related to the presence of abnormal electrical activity, the site of the epileptic focus being of no importance.

In sum, in non-human primates removal of anterior temporal cortex with or without associated ablation of the amygdala causes a range of significant social and emotional changes. Alterations in aggressive behaviours is the most consistently observed emotional disturbance in both man and monkey. However in other respects it is hard to compare human and monkey research. In human research much attention has been directed to the study of the link between psychiatric disorder and temporal lobe epilepsy, whereas in monkey work the social and emotional behaviour of the animal with anterior temporal damage is observed using quantitative ethological indices. Without negating the obvious importance of attempts to relate psychiatric symptoms to temporal lobe dysfunction, it would seem that there is also a place for research where similar methods are applied in the study of emotional disorder for man and monkey. We should therefore attempt to apply direct quantitative observational techniques to the study of the emotional changes experienced by human patients with anterior temporal damage.

### **Subcortical and brainstem structures and emotion**

There are close two-way anatomical connections between the prefrontal and anterior temporal cortices (Nauta, 1962), and damage to either region results in changes in social and emotional behaviour. Though opinions differ about the role of the cingulate gyrus in the control of emotional behaviour, we do know that this cortical region also has anatomical links with frontal and temporal cortex (Brodal, 1981; Baleydier and Maugiere, 1980;). However social

and emotional behaviour is not solely regulated by way of "horizontal" cortico-cortical anatomical pathways. "Vertical" cortical-subcortical neural networks are also involved, and we have seen that damage to anterior temporal cortex which spares the amygdala can produce similar emotional changes to those caused by amygdalar lesions. There are a number of subcortical structures which are richly interconnected, and which also have close anatomical links with the prefrontal cortex, anterior temporal cortex, and cingulate gyrus (Brodal, 1981). For example, the hypothalamus receives projections from the amygdala, septum, ventral tegmentum, central gray, and dorsomedial nucleus of the thalamus (Devito and Smith, 1982). Also the thalamic dorsomedial nucleus is connected with the frontal cortex (Reep, 1984), amygdala (Jones, 1981), and midbrain "limbic" nuclei (Velayos and Suarez, 1982).

**Hypothalamus, amygdala, and septum:** Most lesion studies investigating the emotional changes consequent to subcortical damage have used animal subjects. There are relatively few comparable human investigations, but the human data that is available is consistent with the results from animals. Thus lesions in the hypothalamus, amygdala, and septum have been found to alter aggressive behaviour in animals (Adams, 1979; Chui *et al.*, 1976), and in man (Haugh and Markesbery, 1983; Killeffer and Stern, 1970; Poeck, 1969; Reeves and Plum, 1969). MacLean (1962) elicited penile erection in the squirrel monkey with electrical stimulation of these structures, while Heath (1975) induced sexual experiences with electrical stimulation of the human septum. Blumer and Walker (1975) and Poeck (1969) also associate sexual disturbances with damage to these regions, and Schneider (1977) was able to reduce human hypersexuality using stereotactic hypothalamic lesions. Fear and anxiety have also been induced in man by stimulation of sites in the amygdala, hippocampus, medial hypothalamus (Heath, 1975).

**Thalamus:** In contrast to the considerable data that has been gathered on the hypothalamus, amygdala and septum rather less attention has been devoted to the study of the contribution of more dorsal diencephalic nuclei to emotional processes. Butter and Snyder (1972) found lesions of the dorsomedial nucleus of the thalamus produced social deficits similar to those consequent to orbital-frontal ablation. Their study is consistent with other work where alterations in aggressive behaviour have sometimes been found after damage to this nucleus in animals (Siegal *et al.*, 1972). Penile erection in the squirrel monkey has been induced by electrically stimulating this structure (MacLean, 1962). Stereotaxic lesions in the dorsomedial nucleus have been used to treat psychiatric disorder (Mitchell-Heggs *et al.*, 1976; Spiegel *et al.*, 1951). Further, flattening of affect is a feature of the Korsakoff state (Butters, 1979) which is associated with damage to the dorsomedial nucleus and other dorsal thalamic structures as well as to the mammillary bodies (Brierley, 1977). Poverty of facial expression has also been noted following dorsomedial thalamic lesions in monkeys (Brierley and Beck, 1958), and perhaps in man (Graff-Radford *et al.*, 1985). Lastly, Lobosky *et al.* (1984) and Malamud (1967) describe emotional and psychiatric symptoms associated with colloid cysts of the third ventricle.

**Basal ganglia:** There are also anatomical and functional links between limbic structures and the ventral striatum suggesting that the basal ganglia are involved in the control of emotional behaviour (Lopes da Silva *et al.*, 1984; Heimer *et al.*, 1982). MacLean (1978) found that bilateral lesions in the medial segment of the globus pallidus eliminated or fragmented the trump display of the squirrel monkey. Swanson and Mogenson (1981) report that drinking behaviour is disrupted by lesions of the globus pallidus as well as by hypothalamic damage. These latter authors point out that there are anatomical



links from the ventral tegmental area to the nucleus accumbens which then projects to the globus pallidus. In humans, the basal ganglia have also been implicated in emotional disorders. Depression is a common symptom in patients with Parkinson's disease (Mindham, 1974). A prominent feature of this disease is the depletion of the neurotransmitter dopamine in the nigro-striatal pathways, it is possible that Parkinsonian patients' depression is caused by the depletion of catecholamines in Parkinsonism (Shopsin *et al*, 1974). Nevertheless we must remember that it is also possible to attribute this depression to the patient's psychological reaction to his very distressing disabilities (Warburton, 1967). However one affective change in Parkinsonism that cannot easily be ascribed to a psychosocial cause (a reaction to disability) is the frequently observed flattening of facial expression (Walton, 1981). The reduction in spontaneous facial expression suggests that the nigro-striatal system plays some part in the regulation of facial expression. As we have seen, in monkeys and man a paucity of spontaneous facial expression has been associated with frontal lobe damage. The frontal lobes have close anatomical links with the basal ganglia (Brodal, 1981; Kemp and Powell, 1970) and it is perhaps likely that the frontal cortex modulates the activities of the nigro-striatal system in the production of facial expressions.

**Brainstem:** The midbrain also has a role in the control of emotional behaviour. A range of, albeit abnormal, emotional responses have been elicited in cats after total transection of the cerebrum from the brain stem just caudal to the hypothalamus (Bard and Macht, 1958). Lesions in the midbrain have been found to alter aggressive behaviour in animals (Adams, 1979). Heath (1975) describes fear and anxiety after electrical stimulation of the periaqueductal grey. Midbrain tegmental lesions seriously disrupted the isolation call and species-specific displays in the squirrel monkey (Newman and MacLean, 1982).

More caudal brain stem nuclei are also involved in the production of emotional behaviours. Thus Smith and Flynn (1980a&b) conclude that pontine nuclei make a contribution to aggressive responses, and there is the recent intriguing suggestion that the cerebellum may be involved in emotional expression (Haines *et al.*, 1984; Heath, 1975).

Jurgens (1979), studying the neural systems regulating vocalisations in primates, argues that reflex components of emotional responses are represented in the caudal brain stem nuclei. In his view the midbrain central grey in conjunction with the reticular formation coordinates the activity of the brain stem cranial nerve nuclei to produce the facial, respiratory, and autonomic components of emotional vocalisations.

A similar view to that of Jurgens' has been reached by studies of pathological laughter in man, where patients display involuntary, uncontrollable spasms of laughter in response to mildly humorous stimuli. Thus in his review, Poeck (1967) concludes that laughter is controlled at many levels of the CNS, because pathological laughter has been found subsequent to lesions of the internal capsule including the basal ganglia; or to the substantia nigra; or the caudal hypothalamus and cerebral peduncles; or to bilateral lesions of the pyramidal and extrapyramidal tracts in the caudal brainstem. However while laughter may be controlled at many levels of the CNS, Brown (1967) suggests that pathological laughter is due to a release, or disinhibition, of laughter reflexes represented in the caudal brainstem. This is consistent with Ironside's (1955) observation that it is only in the case of laughter associated with caudal brain stem lesions that the patient's mental state can be at variance with the spasms of laughter they produce. Thus one such patient with both pathological laughter and crying said "I usually become very frustrated and angry at my inability to halt such ridiculous behaviour" (Lieberman and Benson, 1977).

While the study of vocalisations in primates (Jurgens, 1979) and pathological laughter in man are both consistent with the hypothesis that the hindbrain is involved in reflex aspects of emotional responses, a note of caution is appropriate. The role of caudal brainstem and cerebellum in the regulation of emotion has, as yet, received relatively little attention. Therefore with Goldstein (1974) we can conclude that, "At minimum, a reflex centre for these ...affective responses is present in the lower brainstem." (Emphasis added)

### Multiple emotional systems and the classification of emotional disorders

Given that emotional behaviour is the result of activity in an anatomically heterogeneous group of neural structures, which are widely spatially distributed in the brain, we might expect that damage to different structures involved in controlling emotion will produce qualitatively different emotional disorders. Indeed, Butter and Snyder (1972) found that monkeys with orbital-frontal or dorsomedial thalamic lesions showed increased aversion to humans, while monkeys with lesions in the amygdala produced fewer aversive responses post-operatively. Similarly, Franzen and Myers (1973) reported that the emotional sequelae of prefrontal and anterior temporal ablation differed in quantitative terms, with the more severe emotional disturbance following prefrontal removal. Kolb and Nonneman (1974) studied social behaviour in the rat after frontal cortical, or hippocampal, or amygdalar, or septal lesions. They concluded that "Frontolimbic lesions clearly altered social behaviour and, to a large extent, the effects of the different lesions would be dissociated from one another."

If lesions in different sites produce different types of emotional disturbance, we must seek a valid way of classifying and categorising the different types of

emotional disorder that can occur. Two ways of classifying emotional deficit are discussed. Emotional disorders might be placed into ethologically meaningful categories. Alternatively, brain lesions may differentially affect the various components of emotional responses. In the latter case, there may be selective deficits of emotional perception, emotional expression, autonomic reactivity, and inner subjective experience.

**Classification by ethological-functional criteria:** Emotional behaviours can be grouped in ethological terms. For example, maternal behaviours form one class of emotional functions. It has also been argued that there are at least two categories of aggressive behaviour, where offensive-aggressive and defensive-aggressive behaviours are distinct (Panksepp, 1982). Separate neural circuits may mediate expression of each ethological-functional category. Certainly, Smith and Flynn (1980a&b) conclude that "quiet" attack and "affective" attack are controlled by neural circuits which are, to some degree, spatially and anatomically separable. Furthermore, hypothalamic stimulation appears to elicit well established motor responses that are especially significant to the animal and are often species-specific, and Roberts (1969) concludes that these different emotional and motivational functions are mediated by separate neural circuits.

Though it is conceivable that individual categories of emotional behaviour can be selectively disturbed after a focal cerebral lesion, opinions differ on the most accurate way of classifying emotional behaviour. Thus Adams (1979) distinguishes 3 types of aggressive behaviour, while Panksepp (1979) believes these aggressive behaviours can be placed in one of two categories.

**Deficits of component emotional processes:** In an alternative approach to categorising emotional disorders, it is suggested that emotional perception,

emotional expression, autonomic reactivity, and perhaps inner subjective experience can be differentially disturbed consequent to brain damage. Thus, there may be a group of specifically perceptual-emotional disorders, which can lead to inappropriate social behaviour. For example, a prefrontal female may misperceive an approaching dominant male's sexual intent and so responds inappropriately and attacks him. Or the amygdalectomised monkey does not perceive human observer as being a threatening stimulus and so it behaves inappropriately, i.e. it does not retreat in fear. A second class of emotional deficit suggested by this schema is that of motor-emotional disorder. Here, the subject may perceive the stimuli adequately, but is unable to produce an appropriate social response to the stimulus. A motor-emotional disorder may be responsible for the qualitative change in aggression observed by Miller (1976) in his group of rhesus monkeys with ablation of dorsolateral frontal cortex. Whereas normal monkeys make a series of threat gestures before making an aggressive attack, Miller's operates often attacked without giving a warning facial expression. Perhaps these monkeys are able to adequately appreciate their social situation, and were able to make non-emotional facial movements, but they were unable to produce the appropriate warning facial emotional expression. A third general category would be where the emotional deficit simultaneously disturbs more than one component of emotional behaviour. For example, an emotional deficit may not be reducible to either a perceptual-emotional disorder or to a motor-emotional disturbance. A superordinate or "deep" emotional function is impaired so that the perceptual and response deficits caused by disturbance of this superordinate emotional function are different sides of the same coin.

Indeed, human emotional disorders have been classified according to the degree of perceptual-emotional and motor-emotional dysfunction (Hughes *et*

*al.*, 1983; Ross *et al.*, 1981; Ross and Mesulam, 1979). Thus Ross (1981) develops the hypothesis that emotional disorders (the Aprosodias) are analogous to the aphasias. Thus the patient with a motor (Broca's) dysphasia has greatest difficulty in expressing speech, while their ability to comprehend speech is relatively well preserved (Goodglass and Kaplan, 1972). By analogy, in motor aprosodia the patient presents with a flattened and sometimes inappropriate affect, but he largely retains his capacity to comprehend the significance of the emotional behaviours of others. In sensory (Wernicke's) aphasia, comprehension is impaired, while verbal expression is fluent and paraphasic. Similarly the patient with a sensory aprosodia has difficulty understanding emotion expressed by another, and tends to produce inappropriate emotional responses. Ross and Rush (1981) also suggest that other (somatic) components of emotional behaviour can be selectively impaired in focal brain disease. In their study of depression they suggest that "The standard diagnostic criteria often are not applicable since the neurological lesion may distort or even obliterate salient features of depression. Patients actually may deny being depressed or dysphoric, not have a depressive affect, or be totally unaware of abnormal vegetative behaviours." Furthermore, Bauer (1982) describes a patient who appeared to suffer from reduced sexual arousal to visual erotic stimuli, with responses to auditory and tactile erotic stimuli preserved.

### **1.3. Emotional Processes and General Sensori-Motor Functions**

The classification of emotional disorders in terms of a profile of component deficits, requires measures of emotional perception, emotional expression, autonomic reactivity, and inner subjective experience. Of course, such tests may use emotional stimuli and may aim to elicit emotional responses, but this

does not guarantee that these measures are specifically sensitive to emotional dysfunction. For example, impaired performance on the tests of emotional perception might be due to a general perceptual disorder. Put simply, if a test of visual emotional perception were administered to a blind person we would not conclude that his poor score indicated an emotional perceptual disorder. Clearly, to establish an emotional perceptual dysfunction it is necessary to demonstrate that the patient can see the stimulus adequately. Similarly, disorders of emotional expression may be secondary to motor impairments. Thus Izard (1971) describes the social-emotional disturbances in rhesus monkey groups, following bilateral sectioning of the facial nerve in one group member. Therefore, a theoretical model for the neural substrate of emotion, and a related classification system for the emotional sequelae of brain damage is not sufficient on its own to guide empirical research. The relationship between emotional dysfunction and general perceptual and motor disorders must also be considered, and for our present purposes three possible broad categories of deficit are postulated:

1. General sensori-motor deficits
2. Cognitive-emotional deficits
3. Pure emotional deficits

#### General sensori-motor deficits

In three primate studies the social after-effects of prefrontal and temporal pole lesions have been compared with the emotional consequences of more posterior cortical excision (Raleigh and Steklis, 1981; Franzen and Myers, 1973; Butter and Snyder, 1972), and a relative absence of social or emotional



disturbances occurred after posterior excision. For example, Franzen and Myers (1973) investigated the social behaviour of monkeys with bilateral lesions of visual association cortex. On return to their group cages these operated "... immediately rejoined their family groups.....they threatened lower ranking animals and humans, and groomed and were mounted by the adult male." The absence of social or emotional deficit after posterior cortical damage suggests that these cortical regions are not primarily concerned with the regulation of specifically emotional processes. Presumably, such neural regions are primarily concerned with the regulation of general sensori-motor functions.

The general sensori-motor category is defined as comprising a range of sensory, perceptual, motor, and perhaps more complex "cognitive" disorders. It is suggested that removal of striate cortex, somatosensory cortex, or primary motor cortex produces sensori-motor deficits, but does not directly disrupt emotional processes. While the hypothesis that ablation of primary sensori-motor cortex does not directly disrupt emotional processes lacks empirical backing, we have seen that, in three studies, ablation of association temporal neocortex produced no obvious social or emotional sequelae. Also, posterior temporal cortex is specialised for the perception of complex visual stimuli. There is considerable electrophysiological and anatomical evidence showing that visual information passes through striate cortex and is distributed to prestriate and inferotemporal cortex (Zeki, 1978; Gross, 1973; Jones and Powell, 1970). Furthermore single unit electrophysiological studies show that compared with striate neurons, prestriate and inferotemporal neurons are especially responsive to more complex features of the visual stimulus (Perret *et al.*, 1982; Desimone and Gross, 1979; Van Essen and Zeki, 1978). The importance of these regions for complex visual perception is confirmed by the evidence that pattern discrimination impairments occur after prestriate lesions and

concurrent discrimination deficits are found after more anterior inferotemporal lesions (Dean, 1976; Weiskrantz, 1974).

If damage to unimodal visual association cortex does not lead to obvious emotional disturbance, it may also be the case that damage to other unimodal association areas (auditory and somatosensory) similarly results in no significant emotional dysfunction. This question has received very little attention, but Butter and Snyder (1972) contrasted their orbital-frontal monkeys with monkeys who had superior temporal ablations i.e. which encroached on auditory association cortex. They reported no emotional disturbance in this group. Unfortunately there does not appear to be any available evidence concerning possible social and emotional disorder in animals with somatosensory association cortical ablations, nor is there available evidence of emotional changes associated with damage to premotor cortex which appears to be specialised for the control of certain complex movement sequences (Freund and Hummelsheim, 1985; Weinrich *et al*, 1984; Deuel, 1977).

If primary and association cortical lesions do not directly disrupt emotional processes, we are next led to ask whether damage to multimodal cortex can directly affect emotional processes. It is conceivable that, in some instances, damage to multimodal cortex does not directly disrupt emotional behaviour and, certainly, it has already been suggested that specifically emotional deficits may not follow ablation of multimodal premotor cortex. The convergence of sensory data on multimodal cortical zones often occurs after it has passed through several synaptic junctions, so that when it arrives at polymodal cortex it is highly processed and integrated. For example, in parietal multimodal cortex single neurons respond to complex visual and somatosensory stimuli, and one of the complex functions of this region is the maintenance of spatial awareness (Hyvarinen, 1982; Lynch, 1980). However the fact that highly processed sensory

data converges in multimodal cortical zones does not logically imply that such regions must be directly involved in the regulation of emotional processes. It is commonly assumed that emotional meaning is attached to sensory data through contact with limbic processes. Thus Watson *et al.* (1981) have suggested that multimodal parietal cortex does have a role in emotional processes, through its anatomical links with the cingulate gyrus. However the electrophysiological investigations of Rolls *et al.* (1979) did not confirm their conclusion. Future empirical research is required to investigate the hypothesis that, at least some, multimodal regions are primarily concerned with higher order general sensori-motor functions which might be loosely labelled "perceptual-cognitive", or "motor-cognitive", or simply "cognitive".

### Cognitive-emotional deficits

Though some multimodal regions conceivably subserve general sensori-motor functions, subcortical limbic structures receive highly processed sensori-motor information through frontal and anterior temporal multimodal cortical regions (Turner *et al.*, 1980; Pandya and Kuypers, 1969; Jones and Powell, 1970). Thus some of the cortical regions where sensori-motor integrations are at their most complex (i.e. in prefrontal and anterior temporal) are also important for the control of emotional processes. The prevailing view accounting for this rather surprising fact is that prefrontal and anterior temporal regions provide an essential interface between general sensori-motor processes and emotional processes.

Clearly, emotional behaviour often occurs in response to external events. If we assume that certain general sensori-motor neural systems are involved in the initial analyses of external stimuli we must have some physical neural substrate where the results of this general sensori-motor analysis can be

integrated with the animal's emotional states and responses. In short, we need to have certain neural regions where general sensori-motor analyses and emotional functions can meet, or interface. Indeed, Nauta (1971) suggested that the frontal cortex is "... a major mediator of information exchange between the cerebral cortex and the limbic system ...", and recent electrophysiological evidence gives strong support for this view. Thorpe *et al.* (1983) identified individual orbito-frontal neurons responsive to visual presentation of specific food stimuli such as peanuts, banana, and raisins. Some neurons responded to specific aversive stimuli such as a saline containing syringe, but not to another aversive stimulus e.g. an air puffer. For some of the latter neurons, alteration of the reward value of the stimulus produced a corresponding alteration in neuronal response. This responsiveness of orbito-frontal neurons to a specific combination of visual sensory data and information about reward value indicates that this neural region is involved in the creation of an interface between sensori-motor and emotional functions.

Lesion data is also consistent with the suggestion that some cortical regions are important for the creation of an interface between sensori-motor and emotional functions. For example, orbito-frontal ablation produces emotional deficits as well as impairment on several learning tasks such as object alternation (Mishkin *et al.*, 1969). Thorpe *et al.* (1983) argue that the convergence of visual and motivational information on orbito-frontal neurons is a prerequisite for such learning tasks. For some forms of learning to occur it is necessary to attach a motivational meaning to sensory data; one object is to be approached because it has rewarding properties, while another object is to be avoided because aversive motivational properties are attached to it at the interface.

In sum, a common current view is that a sensori-motor-limbic interface in orbito-frontal cortex is important for both emotional behaviour and cognitive/learning skills. It is possible that emotional functions and cognitive/learning functions are mediated by anatomically distinct orbito-frontal interfaces. However it may also be the case that damage at an interface might produce deficits which manifest themselves in **both** cognitive tasks **and** emotional situations. It is possible that there is a category of deficit that is neither cognitive nor emotional, but which could be described as being cognitive-emotional. For example, a perseverative tendency is often a feature of frontal lobe deficits on a variety of cognitive tests administered to man (Milner, 1982; Luria, 1966; Teuber, 1964) and monkey (Rosenkilde, 1979; Brutkowski, 1965). Disinhibition of emotional impulses can also be a consequence of frontal damage. It may be that, in some cases, the perseveration seen in cognitive tasks and emotional disinhibition are different manifestations of an underlying unitary cognitive-emotional deficit resulting from damage to a neural region important for the creation of a sensori-motor-limbic interface.

#### Pure emotional deficits

The final conceivable category to be considered is that of a "pure" emotional disorder. In this case the patient's intellectual, memory, perceptual and motor functions would be completely preserved but the patient's emotional responses would be disturbed in some way. For example, the patient may lose (or gain) sexual interest or he may become more (or less) aggressive. Alternatively, a particular emotional response may occur at the same frequency as before his brain lesion, but there might be qualitative changes in the emotional response. For example, a patient's laughter or anger may assume a "forced", excessively strong or (flat) quality. Of course, it is not sufficient to

conceive of a class of "pure" emotional disorders. It is also necessary to empirically confirm the existence of such syndromes. For Ironside (1955) no cases of "pure" pathological laughter occur with lesions rostral to the midbrain. "At the highest level (anterior hypothalamic, frontal, temporal) emotional lability.....is part of a complex mental disorder shown by excited speech or behaviour and giggling.....Here there may be coexistent deficits of memory, attention and perception."

Nevertheless, in patients with intractable psychiatric disorders attempts have been made to produce strategic lesions of the fronto-limbic systems which selectively relieve the psychiatric emotional symptoms without producing cognitive sequelae. Mitchell-Heggs *et al.* (1976) found a marked reduction in a range of psychiatric symptoms following bilateral stereotaxic lesions in the lower medial quadrant of the frontal lobes, dorsomedial nucleus of the thalamus, and the cingulum. Since there was no evidence of intellectual loss on psychometric tests, this form of psychosurgery suggests that cognitive and emotional anatomical pathways are separate but closely adjacent or overlapping. In this case we might be able to account for Ironside's (1955) inability to find a patient with a supratentorial lesion showing pathological laughter which was uncomplicated by memory, attention or perceptual disorder. If cognitive and emotional pathways are anatomically adjacent we would expect that in most cases patients would suffer from both cognitive and emotional disorders, because in man lesions tend to be large. However this is not always the case, and there may be few patients with a strategically placed cortical or subcortical lesion causing a pure emotional disorder. Such a disorder would be analogous to the dysphasic syndrome of pure word deafness, where ability to perceive spoken words is impaired but all other language skills are preserved (Auerbach *et al.*, 1982).

In sum, it is possible to conceive of a class of pure emotional deficits associated with a supratentorial lesion. In such cases general sensori-motor functions would be completely preserved but a particular type of emotional behaviour would be altered after brain damage. Though the existence of such single cases is not established, they are probably uncommon if they do occur. Generally speaking in supratentorial lesions cognitive and emotional disorders co-occur. This may be because emotional and cognitive circuits are interdigitated or because of the previously described category of deficit which we have labelled "cognitive-emotional".

#### **1.4. Conclusions**

Social and emotional deficits have been consistently observed in man and monkey following damage to prefrontal and anterior temporal cortex, but the role of the cingulate gyrus in the regulation of emotional behaviours is unclear at present. Lesions in several subcortical and brainstem sites are also known to induce emotional disturbances in man and in animals. Though human and animal data both implicated similar structures specialised for the regulation of emotion, species-specific differences in emotional behaviours mean that we must be cautious in the extrapolation of emotional disorders in primates to the human case.

It is also difficult to compare human and monkey work because the methodologies employed in animal and human research are often quite different. Studies of the social-emotional behaviour of brain lesioned monkeys have used reliable quantitative observational measures of the monkey's social interactions or emotional responses. In contrast, studies in man have commonly employed two assessment approaches. In some cases assessment focuses on the inner emotional experience of the patient. The patient is



psychiatrically interviewed about his symptoms and/or he fills in a self-report inventory describing his inner emotional symptoms (Reitan, 1976; Espmark, 1973). Alternatively, patients' emotional symptoms are evaluated through the qualitative, subjective impressions of behavioural change formed by the psychiatric researcher or the patient's spouse (Fordyce *et al.*, 1983; Storey, 1970). To bring human research into line with animal work it is necessary to apply comparable methods in the study of similar behaviours for both human and non-human subjects (Oscar-Berman and Zola-Morgan, 1980). Thus poverty of facial expression after prefrontal ablation has been observed in several primate studies, which used objective reliable observational measures of facial expression. Using quantitative indices Kolb and Milner (1981b) also demonstrated a significant poverty of facial expression in patients with frontal cortical excision for the relief of epilepsy.

It is also suggested that tests of emotional deficit should be based on a classification system, which is derived from an explicit theory about the neural basis of emotion. There is, of course, no established method of classifying emotional disorder. However two possible approaches have been considered. Emotional dysfunction may be subdivided in ethological-functional terms, so that the class of sexual-procreative behaviours can be distinguished from the class of offensive and defensive aggressive behaviours. An alternative way of classifying emotional disorders is to distinguish patients in terms of their profile of scores on a range of measures assessing emotional perception, emotional expression, autonomic reactivity, and subjective response.

A further conclusion is that emotional deficit cannot be investigated without regard to cognitive impairments. Three possible relationships between emotional disorder and cognitive deficit were discussed. In some cases these emotional deficits are secondary to disturbance of general sensori-motor

functions. A class of pure emotional deficits is also conceivable, as is the class of cognitive-emotional disorder. One strategy for investigating these putative relationships between general and sensori-motor and emotional functions, entails the concurrent administration of cognitive and emotional tests. Thus to show that a category of pure emotional disorder exists we would need to find a patient with impairment on one or more emotional measures but no deficit on any test of perceptuo-motor or cognitive function.

## CHAPTER 2

### THE NEURAL SUBSTRATE OF EMOTION: THE INTER-HEMISPHERIC DIMENSION

Until recently research into the neural substrate of emotion has concentrated on the intra-hemispheric dimension, and in the first chapter we have discussed the evidence indicating that emotional processes are regulated by prefrontal and anterior temporal cortical-subcortical-brainstem neural systems. However it is also possible that the right and left hemispheres each play a different role in the control of emotional behaviour. Indeed, over the last two decades there has been intense interest in this latter possibility, and current theories about the lateralisation of emotional processes can be grouped under three main headings. First, it has been suggested that the right hemisphere is specialised for the control of all emotional processes. A second possibility is that neither hemisphere is dominant for emotion. The right-left differences in emotional test performance can be explained by well established right-left cognitive functional asymmetries. According to the third theory, each hemisphere is specialised for the control of different emotions. In its current form, this hypothesis states that the left hemisphere is specialised for positive emotions, and the right hemisphere is specialised for negative emotion.

#### **2.1. Right Hemisphere Dominance for Emotion**

##### **Dominance for emotional perception**

Several studies of patients with unilateral brain injury have suggested a right hemisphere dominance for emotional perception. Thus, patients with right hemisphere disease are particularly poor at identifying facially expressed emotion (Etcoff, 1984; Kolb and Taylor, 1981; Cicone *et al*, 1980; DeKosky *et al*,

1980). Though these studies relied on still photographs of emotionally expressive faces, Benowitz *et al.* (1983) used filmed emotional faces and also found that the right hemisphere was specialised for the perception of facially expressed emotion. However, Gazzaniga *et al.* (1975) could not demonstrate a right hemisphere advantage when split brain subjects were presented with emotional faces, and Goldblum (1980) only found a non-significant trend for greater right hemisphere deficit on a test of facial emotional perception.

Heilman *et al.* (1975) presented patients with sentences spoken in an angry, sad, happy or emotionally neutral tone of voice. Compared to patients with left hemisphere injury, a group with right hemisphere lesions were significantly less able to judge affect on the basis of vocal tone. Similar results have been reported since this original investigation (Heilman *et al.*, 1984; Hughes *et al.*, 1983; Tucker *et al.*, 1977). Unfortunately these studies were vulnerable to uncontrolled biasing influences, because group sizes were small and subjects were selected on the basis of their neuropsychological deficits. In an investigation using a less highly selected sample, Schlanger *et al.* (1976) found no evidence of greater right hemisphere impairment on a test of perception of emotionally toned sentences.

Other investigators have used stimuli where emotion was expressed in verbal-semantic content. Brownell *et al.* (1984) concluded that patients with unilateral right hemisphere disease had particular difficulty in understanding the emotional significance of words. Wechsler (1973) also found a right hemisphere deficit when patients were required to recall an emotionally charged narrative text. However Kolb and Taylor (1981) found a greater left hemisphere impairment on a task where patients had to name the emotion expressed by written text.

Studies of normal non-brain injured subjects have also indicated that the right hemisphere may be dominant for emotional processing (Ley and Bryden, 1979; Lavadas *et al.*, 1980; and Suberi and McKeever, 1977). For example, in a tachistoscopic study Ley and Bryden (1979) presented emotional faces to the right or left visual half-field. Subjects were significantly quicker at perceiving the emotion expressed when the faces were flashed in the left visual field. In addition, Graves *et al.* (1981) concluded that the right hemisphere makes a unique contribution to the perception of emotional words, but Strauss (1983) was unable to replicate this finding.

### Dominance for emotional expression

In two influential studies, Gainotti (1972 and 1969) observed patients' reactions to failure while he administered a battery of cognitive tests. He identified two types of reaction. Patients with left hemisphere disease more frequently displayed the catastrophic reaction (Goldstein, 1942). When confronted with failure such patients become anxious, or tearful, or aggressive. In contrast, patients with right unilateral lesions more often showed an indifference reaction (Denny-Brown *et al.*, 1952). When these patients fail test items they appear unconcerned and tend to joke, minimise or deny their deficits. Somewhat similar findings have been reported from several studies on the spontaneous reactions of patients receiving unilateral intracarotid sodium amytal injections. In some of these investigations, left intracarotid injection tended to induce a depressive-catastrophic reaction, while a euphoric reaction was more frequent after anaesthesia of the right hemisphere (Rossi and Rosadini, 1967). Buck and Duffy (1980) reported that right hemisphere lesioned patients were significantly less emotionally expressive than left lesioned patients when watching emotionally evocative films. Bruyer (1981) found that

the voluntary facial emotional expressions of patients with right brain damage were less intense than the facial emotional expressions of a group with left hemisphere lesions.

A number of studies using normal subjects also suggest a right hemispheric specialisation for emotional expression. Thus Moscovitch and Olds (1982), Borod and Caron (1980) and Campbell (1978) found that the left half of the face is more expressive emotionally. Sackheim *et al.* (1978) concluded that emotion is expressed more intensely on the left hemiface by constructing symmetrical photographs made up entirely from the right or the left hemiface. They used photographs of faces of people posing a range of emotions. Each photograph and its mirror reversal was split down the vertical midline. Then by joining the right half of the original photograph to the left half of the mirror image they produced a symmetrical composite photograph of the face which had been wholly made up of the right half of the face. Left face composites were constructed in a similar fashion. The left face and right face composites were then rated for emotional expressiveness by independent judges. In this study, and in the majority of similar studies (Dopson *et al.*, 1984; Rubin and Rubin, 1980), the left-sided composites expressed emotions more intensely than right-sided composites. Nevertheless, Ekman *et al.* (1981) found no right-left asymmetries for spontaneous facial expression in normals, and in a recent review Thompson (1985) emphasised the inconsistencies in the currently available evidence.

In sum, the view that the right hemisphere is dominant for emotional functions is supported by a fair body of research data using brain-injured and normal subjects. The data are reasonably consistent for the perception of facially expressed emotion, but there are also indications that the right hemisphere is specialised for the perception of emotion in tone of voice, as

well as for voluntary and spontaneous facial expressions. Unfortunately, in much of this work the relationship between emotional asymmetries and perceptuo-motor hemispheric asymmetries is undetermined. However Weintraub *et al.* (1981) suggest that right lesioned patients' difficulty in perceiving emotion expressed in tone of voice is merely one manifestation of a primary inability to perceive complex intonational patterns. In other words, the deficit in perception of emotional tone may be secondary to a primary auditory perceptual impairment. Put more generally, in most tests of emotional function the patient has to perceive stimuli and/or make some response. That is to say, most tests of emotional function require effective performance of perceptual, motor and cognitive skills. It is conceivable that some (or indeed all) differences between right and left lesion groups on emotional tests are merely secondary manifestations of primary perceptuo-motor and cognitive asymmetries.

## 2.2. The Relationship Between Emotional and Cognitive Hemispheric Asymmetries

We will next briefly review the evidence for differential right-left perceptuo-motor and cognitive specialisations. Using this as a base, we can then examine the possibility that emotional hemispheric asymmetries are secondary manifestations of primary perceptuo-motor and cognitive deficits.

### General perceptuo-motor and cognitive hemispheric asymmetries

Surveys of patients with unilateral lesions have consistently found an association between dysphasia and left hemisphere injury (e.g. Luria, 1970; Russell and Espir, 1961). Patients with left hemisphere damage tend to do less well on Verbal IQ measures such as the Mill Hill Vocabulary Test (Newcombe,



1969), and verbal memory deficits are associated with left temporal lesions (Milner, 1971 & 1958). There is also considerable evidence indicating that the left hemisphere is specialised for certain motor skills (De Renzi *et al.*, 1983), and most patients with ideomotor and ideational apraxia suffer from left hemispheric injury (De Renzi *et al.*, 1980; Heilman, 1979; De Renzi *et al.*, 1968).

Right hemisphere functional specialisations are also well established. Zangwill and his colleagues in an influential series of reports described a syndrome of "unilateral spatial agnosia" which was particularly associated with injury to the posterior region of the right hemisphere (Ettlinger *et al.*, 1957; McFie *et al.*, 1950; Patterson and Zangwill, 1945 and 1944). Their patients suffered from a range of perceptuo-motor and spatial deficits including unilateral visual neglect, spatial disorientation, constructional apraxia and dressing apraxia. Several studies have confirmed the increased incidence and severity of unilateral visual neglect after right hemispheric damage (Colombo *et al.*, 1978; Albert, 1973; Faglioni *et al.*, 1971; Gainotti, 1968). Right hemisphere lesions are associated with selective deficits in face identity matching (Hamsher *et al.*, 1979; Meadows, 1974; Yin, 1970; Benton and Allen, 1968; Warrington and James, 1967); perception of colour hues (Scotti and Spinnler, 1970) and of objects at unusual angles (Warrington and Taylor, 1978); perception of unfamiliar sounds (Vignolo, 1982; Faglioni *et al.*, 1969) and musical rhythms (Milner, 1962); and tactile perception (Milner and Taylor, 1972; Newcombe, 1969; De Renzi and Scotti, 1969).

In fact throughout the 1960's and 1970's many studies employing extremely diverse methodologies have firmly established hemispheric functional asymmetries. There have been investigations of patients with commissurotomies for the control of epilepsy (Sperry *et al.*, 1969; Levy *et al.*, 1972), dichotic and tachistoscopic investigations of non-brain injured normal

subjects (Bradshaw and Nettleton, 1981), EEG studies (Heilman and Van Den Abell, 1980), cerebral blood flow studies (Risberg *et al.*, 1975) and ECT studies (Berent, 1977), all indicating the presence of right-left asymmetries. We also know that functional asymmetries are not confined to the cortex, but occurs in cortical-subcortical neural systems. Thus Milner (1965) found that the degree of spatial memory disturbance was directly proportional to the extent of right hippocampal removal. Naeser *et al.* (1982) and Mohr *et al.* (1975) report dysphasias associated with lesions in the left lesions in the left putamen and left thalamus, respectively. Damage to (or electrical stimulation of) the left thalamus has been associated with impairment on tasks employing verbal stimuli while right thalamotomy (or stimulation) produces deficits on tasks employing non-verbalisable stimuli (Ojemann, 1977; Vilkki and Laitinen, 1976 and 1974; Fedio and Van Buren, 1975).

In spite of the agreement about the presence of hemispheric functional asymmetries, consensus on the definition and explanation of these specialisations is lacking. It is commonly said that the left hemisphere specialises in verbal functions and the right specialises in nonverbal functions. However Bradshaw and Nettleton (1981) examine the problems with the verbal/nonverbal distinction, and review alternative ways of categorising the differences between right and left hemisphere functions. No attempt will be made to exhaustively catalogue all the suggestions that have been made to date, but it is necessary to describe one widely discussed explanation for hemispheric functional asymmetries.

Hecaen and Angelergues (1963) made the quasi-physiological suggestion that the right hemisphere is diffusely organised while the left hemisphere is focally organised. These workers found that lesions of the right parieto-temporal, parieto-occipital and parieto-temporo-occipital regions all

produced the typical right lesion deficits of dressing dyspraxia, unilateral neglect and dyscalculia. In contrast there was a **more specific** association between deficit and lesion site after left sided injury. For example, colour agnosia largely followed a lesion of the occipital lobe, but ideomotor apraxia was not associated with damage confined to this region. These authors concluded that different functions are **diffusely** represented in the right posterior brain whereas functions are more **focally** represented in the left posterior brain. They further suggested that the considerable degree of equipotentiality in the right posterior brain lends itself to the synthesis of divergent sensory input. They hypothesise that the (diffuse) right hemisphere is primarily concerned with global analysis of the sensory environment. At the neurophysiological level, processing is more primitive and so immediateness and affective value is retained. Conversely, the organisation of the left posterior brain, where different functions are located in relatively discrete and circumscribed areas, is more suited to a categorical analysis of sensory data. They suggest that the combination of focal organisation and language skills makes the left hemisphere specially equipped for the conceptual elaboration of sensory data by means of language. Though it has its supporters (Bradshaw and Nettleton, 1981; Semmes, 1968), the focal/diffuse dichotomy also has its critics (Newcombe, 1974).

#### Emotional asymmetries as secondary manifestations of primary cognitive hemispheric asymmetries

In left hemisphere patients, Schlanger *et al.* (1976) found an association between severity of dysphasia and ability to identify emotion expressed through tone of voice. Gardner *et al.* (1975) presented captioned and uncaptioned humorous cartoons to right and left injured patients. They similarly

found that left hemisphere dysphasic patients had particular difficulties in understanding the cartoons which had a written caption. Seron *et al.* (1982) also reached this conclusion, and Kolb and Taylor (1981) reported that their left lesion group was selectively impaired where patients had to name the emotion expressed in written sentences. Therefore, although the relationship between dysphasia and deficits of emotional perception has, so far, received relatively little attention (Feyereisen and Seron, 1982), the impaired performance of left hemisphere patients on verbal emotional tests is probably, in part, due to language disturbances.

The possible dependence of emotional perception on right hemisphere perceptual-cognitive functions has also been considered. Strauss and Moscovitch (1981) and Ley and Bryden (1979) tachistoscopically presented pairs of emotional faces to normal subjects. Sometimes subjects had to say whether the stimulus pair was of the same person (identity matching), on other occasions the subject had to decide whether the faces expressed the same emotion (emotional matching). Both studies found a left visual field advantage for facial identity and emotional matching. However, using analysis of covariance, Ley and Bryden (1979) concluded that facial identity and facial emotional recognition were separate and dissociable right hemispheric functions. Similar results have also been found in patients with unilateral lesions. Dekosky *et al.* (1980) found that the right hemisphere group had particular difficulty in perceiving emotional faces. Though their right lesioned group also showed greater impairment on a prosopagnosia screening test, Dekosky *et al.* (1980), Bowers and Heilman (1984), and Etcoff (1984) described single cases where identity recognition was preserved but emotional perception was impaired. Etcoff (1984) also found the converse pattern, where emotional perception was impaired but facial identity perception was relatively preserved.

Therefore, deficits on tests of perception of facially expressed emotion may be partly secondary to a general (perceptual) inability to process facial stimuli. However it is possible that perception of emotional faces is a special skill which can be selectively impaired in focal brain disease.

There appear to be two possible explanations for the right-left differences on emotional tests. One possibility is that all hemispheric emotional asymmetries may be secondary manifestations of primary perceptuo-motor, language, or cognitive asymmetries. Alternatively, some emotional asymmetries are reducible to right-left cognitive differences, but the right hemisphere may also be dominant for certain emotional functions. In this latter case, cognition and emotion may be related in two ways and, in the previous chapter, pure emotional deficits and irreducible cognitive-emotional deficits were theoretically distinguished. Thus a patient would be said to have a pure emotional deficit if he was unable to appreciate the emotional significance of facial expressions, while facial perception was normal in all other respects. However in an irreducibly cognitive-emotional disorder emotional and cognitive deficits are indissociable. There are a number of theories suggesting right hemisphere emotional deficits are of this type, and two such theories can now be briefly mentioned.

#### Cognitive-emotional asymmetries

Gainotti (1972) found a strong association between unilateral neglect and the emotional indifference reaction. Furthermore, both symptoms were associated with lesions in the right hemisphere, which is diffusely organised, according to Hecaen and Angelergues (1963). Gainotti speculated that the diffuse right hemisphere synthesis of divergent sensory input may be necessary for (a) our vivid and immediate awareness of our surrounding

sensory environment, and (b) awareness of the emotional significance of sensory input. Therefore a right hemisphere lesion disrupts this immediate and affective awareness of the external environment, and consequently the patient displays neglect and emotional indifference. In short, Gainotti (1972) hypothesised that neglect and the indifference reaction are different manifestations of a unitary disorder of the diffusely organised right hemisphere functions.

Heilman and his associates suggest an attentional/emotional disorder underlies a link between left unilateral neglect and emotional indifference (Heilman *et al.*, 1978). They suggest that the reticular formation directs spatial attention in response to (a) external sensory input, and (b) "limbic" emotional responses and motivational states (Watson, 1981). Thus direction of spatial attention requires convergence of sensory, limbic and reticular processes. Comparative data indicates that the necessary convergence occurs through frontal, parietal and cingulate multimodal cortex (Watson and Heilman, 1979; Watson *et al.*, 1978; Watson *et al.*, 1974; Watson *et al.*, 1973). Damage to such multimodal cortical zones induces deficits which are simultaneously emotional, and attentional, and perceptuo-motor. Furthermore, these attentional/emotional functions are lateralised in the right hemisphere in man (Heilman and Valenstein, 1981; Heilman and Van Den Abell, 1979). Therefore a right hemisphere lesion leads to a combination of neglect and emotional indifference.

### **2.3. Hypothesis of Differential Lateralisation of Emotion**

It has been suggested that the left hemisphere is more involved in the perception of positive emotions (Natale *et al.*, 1983) while the right hemisphere plays the greater role in the perception of negative affect (Ahern and Schwartz,

1979; Reuter-Lorenz and Davidson, 1981). Dimond and Farrington (1977) and Dimond *et al.* (1976) employed an ingenious contact lens technique which allowed them to project emotionally evocative films to either the right or the left hemispheres of normal subjects. It was found that an (emotionally negative) film of a surgical operation produced the greater emotional response when presented to the right hemisphere. Conversely, an (emotionally positive) comedy film produced the stronger emotional response (as measured by subjective report and by heart rate) when it was presented to the left hemisphere.

In a study of non-brain damaged subjects, Sackheim and Gur (1978) suggested that that left and right hemispheres might also be differently specialised for the expression of emotion. Furthermore, Schwartz *et al.* (1979) measured EMG in the left and right zygomatic facial muscles during tasks designed to elicit emotional responses. They found a positive emotional response elicited relatively greater right muscle activity than left muscle activity. Conversely, a negative emotional response produced relatively more left muscle activity.

However Hirschman and Safer (1982) concluded that the arguments in support of the differential lateralisation of emotion using normal subjects is weak, as there are a number of studies producing evidence which directly contradicts this hypothesis. These investigations have found the left visual field to be more effective in the perception of **all** emotions – positive as well as negative (Duda and Brown, 1984; Dopson *et al.*, 1984; Hirschman and Safer, 1982; Strauss and Moscovitch, 1981; Strauss and Kaplan, 1980).

Evidence from brain injured subjects has also been used to support the differential lateralisation of emotion hypothesis. Thus in a literature review



Sackheim *et al.* (1982) concluded that pathological laughter was predominantly associated with right hemisphere lesions, while pathological crying was associated with left sided lesions. They hypothesised that positive emotional systems represented in the left hemisphere, and negative emotional systems of the right hemisphere are mutually inhibitory. Thus a left hemisphere lesion induces pathological crying through the disinhibition of the negative emotional processes in the opposite hemisphere. Conversely, the increased incidence of pathological laughter after right hemisphere damage is due to the disinhibition of left hemisphere positive emotional processes.

However Sackheim *et al.* (1982) found an opposite pattern for epilepsy. Laughter occurring during epileptic seizures was associated with **left** hemisphere epileptic foci, while crying was linked with **right** sided foci. Sackheim *et al.* argue that these apparently contradictory findings are in fact quite consistent if the nature of the lesion is taken into account. On one hand, they suggest that destructive lesions result in disinhibition of contralateral regions. On the other hand, they suppose that abnormal electrical activity in epilepsy activates the emotional systems of its own hemisphere, and this also leads to an increased inhibition of contralateral emotional systems. Thus, a right hemisphere epileptogenic focus excites the negative emotional systems represented in the right hemisphere, and suppresses left hemisphere positive emotional systems.

Sackheim *et al.*'s (1982) explanation for the differing effects of destructive and epileptogenic lesions is certainly logically consistent. However their arguments are far from being compelling. While it may be the case that abnormal electrical cerebral activity can under certain circumstances enhance or trigger responses (Adams, 1979; Bear, 1979) in other circumstances abnormal electrical activity appears to mimic the effect of a destructive lesion (Stamm

and Rosen, 1973; Penfield and Roberts, 1959). Clearly until further evidence is forthcoming it would be unwise to presume that pathological emotion in epilepsy is always due to activation of emotional systems ipsilateral to the epileptic focus. In any case, when Strauss *et al.* (1983) examined films of patients undergoing epileptic seizures, they found no consistent relationship between side of seizure onset and type of spontaneous facial expression.

In sum, the available data on normals and brain injured subjects does not strongly support the view that positive and negative emotional systems are lateralised in the left and right hemispheres, respectively. Nevertheless, cognitive functions are asymmetrically represented in the cerebral hemispheres, and there is evidence that neurochemical pathways are sometimes lateralised (Oke *et al.*, 1978). It is therefore possible that each hemisphere is responsible for different types of emotion. What these different emotional types might be are clearly a suitable subject for empirical investigation.

#### **2.4. General Conclusions and Methodological Implications**

Emotional functions may be asymmetrically represented in the cerebral hemispheres. A fair body of evidence can be cited in support of the hypothesis that the right hemisphere is dominant for all emotional functions. In contrast, there is only limited empirical backing for the alternative current view that positive and negative emotional processes are lateralised in the right and left hemispheres, respectively. However in studies of emotional disorder in neurological patients, dysphasia and perceptual disturbances are probably (at least partially) responsible for deficit on emotional tests, but the influences of emotional and cognitive deficit are often confounded. Nevertheless, when emotional and cognitive tests have been concurrently administered, it has

sometimes been possible to argue that cognitive and emotional deficits are dissociable.

Serious interest in the possibility that emotional functions are lateralised has developed over the last two decades. In contrast, the link between emotional processes and anterior neural systems has been discussed for over a century. However, investigators have tended to study **either** the intra-hemispheric dimension **or** the inter-hemispheric dimension. This is unfortunate, as it is possible that the influences of these two dimensions combine. For example, if the right hemisphere is dominant for emotional processes, and the frontal lobes are specialised for the regulation of emotion, we might predict that right frontal damage is particularly liable to cause emotional disorder. Certainly, Lishman (1968) found affective or behavioural disorders were most frequent after right frontal lesions. Alternatively, if left hemisphere damage is particularly liable to induce the catastrophic reaction, then we might find that the catastrophic response is most marked in patients with left anterior damage. Indeed, Gainotti (1972) suggested that the aggressive responses are disinhibited in dysphasics with left anterior lesions. Furthermore, Robinson and Benson (1981) found depression was particularly common after left anterior damage.

Investigators may tend to focus on either the inter- or intra-hemispheric dimension to emotional neural systems, but there are methodological issues common to both lines of research. First, patient samples are often highly selected, or abnormal in some respect. In the investigation of the intra-hemispheric dimension, the reports about the emotional changes associated with prefrontal leucotomy or stereotaxic amygdectomy are a valuable information source. However the patients undergoing these psychosurgical procedures are always psychiatrically disturbed prior to

operation. Therefore it is not possible to distinguish premorbid psychiatric emotional disorder from the post-operative emotional consequences of the brain lesion. Some studies of the inter-hemispheric aspect of emotional processes are also vulnerable to selection bias. For example, in their important investigation of auditory affective agnosia, Heilman *et al.* (1975) only recruited patients with neglect or dysphasia in association with a right or left hemispheric lesion. Their intriguing conclusion that auditory affective agnosia is particularly closely associated with right hemisphere lesions and left sided neglect may, in fact, be an artifactual consequence of their using selected patients. Indeed, when a similar test of emotional perception was administered to a less highly selected series of patients with a unilateral lesion, no hemispheric differences were found (Schlanger *et al.*, 1976). Clearly, the interpretation of experimental findings can be greatly simplified when patients who have no premorbid psychiatric disorders are recruited. Also, if possible, consecutive series of patients series should be employed, where selection is based solely on lesion variables (location and aetiology), while the nature of the patient's neuropsychological deficit is disregarded during selection.

Secondly, the reliability and validity of emotional measures is not assessed in some studies of anterior and lateralised neural systems controlling emotional responses. For example, evidence that frontal lobe damage is associated with emotional disorder is largely dependent on the clinical impressions of neurologists and psychiatrists. Similarly, Gainotti (1972 and 1969) observed the emotional reactions of patients with unilateral lesions during neuropsychological testing. His clinical observations were not matched with those of an independent judge. However in the absence of interjudge reliability data it is always possible that the clinician's observations are biased and distorted by his own particular subjective opinions.

A third limitation in studies of the neural substrate of emotion is that different aspects of emotional disturbances are often considered separately and in isolation. Studies have focused on emotional perception (Dekosky *et al.*, 1980; Cicone *et al.*, 1980), or emotional expression (Bruyer, 1981; Buck and Duffy, 1980), or autonomic response (Morrow *et al.*, 1981), or self-report of subjective experience (Gasparrini *et al.*, 1978; Folstein *et al.*, 1977). Furthermore, investigators tend to collect information largely from the relative (Weddell *et al.*, 1980), or the patient (Heilman *et al.*, 1983; Ross, 1981). This "fragmented" approach to the study of emotional deficit prevents us from considering the relationships between the components of emotional behaviour. For example, it is not clear whether deficits in making voluntary facial expression have any connection with the emotional disorders observed by the patient's spouse. We might also ask whether if deficits in emotional expression are dissociable from deficits of emotional perception. The degree of association between disturbances of autonomic reactivity and pathological emotional expression could also be evaluated. In fact, there are several interesting issues that can be investigated if multiple measures are made of emotional behaviours.

## CHAPTER 3

### METHODS AND PROCEDURES

#### **3.1. Aims of the Present Investigation**

It has been suggested that emotional behaviour results from the coordinated activity of several component emotional processes. These component processes may be mediated by anatomically differentiated neural circuits. To investigate this possibility, tests of emotional perception, and emotional expression, and subjective experience, and autonomic reactivity are administered to patients with focal cerebral lesions. The correlation between different indices of emotional function was investigated. If the hypothesis that emotional behaviour is mediated by several neural systems is true, then deficit on at least some emotional measures will be dissociable.

However emotional and cognitive deficits co-occur after brain damage, and three theoretically possible relationships between emotional and cognitive deficit were specified. First, emotional deficit may be secondary to primary perceptuo-motor or cognitive impairment. Secondly, pure emotional deficits may exist, where cognitive and emotional impairment are independent and dissociable. Thirdly, a class of irreducibly cognitive-emotional deficits is conceivable. To investigate these three theoretical possibilities, tests of perceptual, motor, language and cognitive function were also administered.

Animal and human studies suggest a special role for the frontal lobes in the control of emotional behaviour. To investigate this possibility the battery of emotional and cognitive tests was administered to patients with either anterior, or posterior lesions.

Given the considerable recent interest in the possibility that emotional functions are asymmetrically lateralised in the cerebral hemispheres. In the present study, tests scores of patients with right and left hemisphere lesions were also contrasted. Three hypotheses were considered. First, the right hemisphere may be dominant for all emotional processes. Secondly, positive and negative emotional processes may be lateralised in the left and right hemispheres, respectively. Thirdly, emotional processes may not be asymmetrically represented in the cerebral hemispheres, as right-left differences in emotional test scores may be secondary to asymmetries of perceptuo-motor and cognitive functions.

### 3.2. Subjects

Forty-eight patients with single localised cerebral lesions verified on Computerised Tomography (CT) were compared with 10 non-brain damaged control patients. In the brain damaged group there were 26 male and 22 female patients. 6 patients had a meningioma, 31 patients had a glioma, and 3 cases had a single cerebral metastasis. Though the majority of our subjects had a histologically verified cerebral tumour there were also 3 stroke patients; 2 with spontaneous intracerebral haemorrhage unassociated with subarachnoid haemorrhage and 1 with a discrete ischaemic cerebral infarct. 3 patients had had an arteriovenous malformation excised. Lastly, 2 patients had an extrinsic lesion pressing upwards on the hypothalamus and adjacent structures, one of these had a suprasellar meningioma and the other had a craniopharyngioma. Patients were excluded from study if they had previously suffered from a serious psychiatric illness. Patients were also excluded if there was evidence of possible previous generalised brain damage e.g. due to alcoholism or closed head injury.



There is now empirical support for the long held view that in the period following damage to one neural structure other neural regions can compensate for certain of the functions lost (Goldberger, 1974). To minimise the extent of such functional reorganisation it has been suggested that patients should be tested within the acute phase of their illness (Tognolo and Vignolo, 1980). Thus only patients with a first stroke who could be seen no more than 60 days after onset were included. In regard to tumour cases, those with tumour recurrence were excluded. In most cases symptom history was less than 2 months, but for 13 patients some neurological symptoms such as epilepsy had first appeared up to 4 years previously. Patients were tested as early as possible in the post-operative period, as soon as they were able to respond appropriately to the test instructions (in 92% of cases testing was completed within 2.5 weeks). All extrinsic tumours had been excised, except in the single patient with a craniopharyngioma which was debulked. In the case of intrinsic tumour, 8 patients had a frontal lobectomy, 3 had a temporal lobectomy, and in 3 cases one occipital lobe was excised. Ten patients had a decompression with subtotal tumour removal, while 7 patients only had a burr-hole biopsy. Six tumour patients were left unoperated. As patients were seen during the acute phase of their illness, most were taking steroid medication at time of testing.

This lesion group was compared with a non-brain injured control group of 10 patients whose spinal lesion was below the C3 level. The mean age for the control (48.8 years, SD=14.7), and brain damaged (50.2, SD=11.6) groups did not differ significantly ( $t=0.40$ ;  $df=55$ ;  $p=0.7$ ). There were 6 male and 4 female controls, and the proportion of males to females was similar in the brain damaged and control groups ( $\chi^2=0.01$ ,  $p=1.0$ ). Table 1 shows that the controls may be from a slightly higher social class (OPCS, 1970) than the lesion subjects, but this difference was far from significant (Mann-Whitney  $U=193$ ;

$p=0.97$ ). Most patients approached willingly agreed to participate in the study, but 4 controls, 2 left hemisphere, 1 left posterior, and one right hemisphere patient refused to be tested.

Social Class	I	II	III	IV
Lesion group	4	12	26	6
Controls	2	2	6	0

Table 1

Social class distribution in the  
brain damaged and control groups

To assess handedness, subjects were asked to state the preferred hand when writing, combing hair, throwing a ball, brushing teeth, and when holding a knife during a meal. If subjects indicated that the right hand was preferred for these 5 activities, they were asked whether they used the left hand in preference for any other task. One patient appeared to be strongly left handed, and 6 were of mixed handedness. The other 41 patients reported themselves to be strongly right handed.

### 3.3. Lesion Location

Patients were scanned with one of three machines; the GE 8800, with a 320\*320 pixel matrix; the EMI 5005, with a 160\*160 pixel matrix; and the EMI 1010, with a 160\*160 pixel matrix. In tumour patients lesion boundaries were determined with the enhanced scans. Though all scans were recent (less than 3 weeks previously), in 30 cases lesion boundaries were determined using

preoperative scans, whereas the patient was tested after tumour debulking and/or lobectomy. Pre-operative scans were also used for a further 7 tumour patients who were assessed after a burr-hole biopsy. There were 6 patients whose tumour was inoperable, or who were seen prior to operation. Finally, post-operative or post-stroke scans were used for the 5 patients with vascular lesions.

### Lesion boundaries

Ratings of lesion boundaries on CT were made conservatively. Inference was kept to a minimum and only regions which were quite clearly of high or low radiodensity were included within the lesion boundaries. The area of abnormal density regions in each CT cut was measured using a transparent millimetric grid which was placed over the total lesion outline. An estimate of total lesion volume was obtained by summing the abnormal density areas in all cuts. This was then multiplied by (a) the thickness of the CT slices and also (b) a magnification factor to scale up the CT volume to life size. Gliomas were the most common lesion type, and were also the most difficult to rate. To establish that it was possible to reliably outline the abnormal density regions in the CT scans of glioma patients two judges separately and independently rated 10 scans. The total lesion boundaries of the two judges were generally congruent on visual inspection, and interjudge agreement about estimated total lesion volume was high (Pearsons  $r=0.94$ ).

### Lesion mapping

The boundaries of the lesion were mapped onto a standard lateral diagram according to the procedures described by Mazzocchi and Vignolo (1978) and

Luzzatti *et al.* (1979). The standard lateral sketch used was taken from Mazzocchi and Vignolo (1978) and was successively xerox enlarged to produce 8 diagrams ranging from 18.6cm to 16.2cm in a-p diameter. The diagram corresponding to the patient's actual brain size was selected by measuring the maximum a-p diameter on the CT images. This diameter was converted to life size by multiplying by a constant magnification factor, which had a different value for each scanner.

To determine the angle of the CT sections, identifiable landmarks on the CT images were related to the corresponding landmarks drawn on the standard lateral diagram. These corresponding landmarks include (a) bones and calcified regions such as sella turcica with posterior clinoid processes or calcified pineal gland; (b) cerebrospinal fluid spaces such as mesencephalic cisterns, and the ventricles. A transparent overlay of parallel lines 1 cm apart (corresponding to CT slice thickness of 1 cm) was superimposed on the diagram (Fig. 1a). The overlay was then oriented with respect to the standard diagram so that the landmarks shown on the first CT slice were also represented within the space between the first pair of parallel lines. This procedure was then repeated for higher cuts. Because of distortions due to mass effect and individual variation in brain morphology, landmarks on the CT slices did not always fall exactly within the appropriate slice on the standard diagram. However the orientation of the overlay was adjusted to minimise such discrepancies. The mean difference in estimated angle of CT slices between the two raters was 3.6 degrees (SD=2.34 degrees).

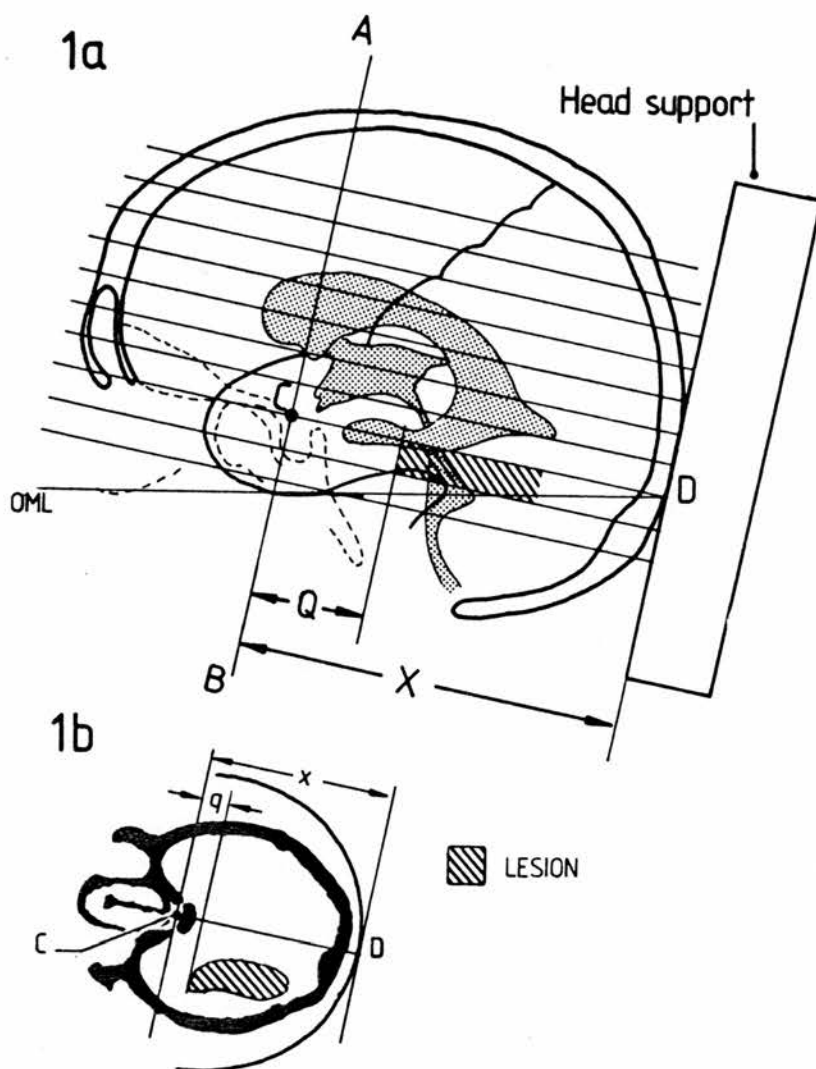


Figure 1

(a) Standard lateral diagram with overlay of parallel lines 1 cm apart, to represent CT slices. Reference axis (AB) passes through midpoint between anterior clinoid processes at C. The axis is at a constant distance CD (X cms) from the head support. The anterior boundary of the lesion is shown at Q cms from the reference axis. (b) CT slice containing reference point C, which is midway between the tips of the anterior clinoid processes. The reference axis passes through all CT cuts at a distance x cms from the head support. The anterior and posterior boundaries of the lesion are defined with respect to the reference axis. In this example the anterior lesion boundary starts q cms behind the reference point at C. (See text for fuller details.)



To map the lesion boundaries onto the standard lateral diagram, the anterior-posterior limits of the lesion boundaries were defined in terms of their distance from a reference axis constructed to pass through corresponding points of the standard lateral diagram and each CT image. The axis is projected through a single bony reference point identifiable on both the standard diagram and on one of the CT images. (In Figure 1a&b the point C between the tips of the anterior clinoid processes provides such a common reference point.) The reference axis (AB) is at right angles to the CT slices and is parallel to the head support, which is visualised as a semi-circular line on each CT image. This is shown in Figure 1a, where the reference axis (AB) is at a constant distance of X cms from the head support of the CT scanner. Figure 1b shows the reference point (C) is x cms from the head rest. As the reference axis is parallel to the head rest, it can be identified on every CT image because it passes through a point x cms from the head rest.

In figure 1b the distance between the reference axis and the anterior most point of the lesion is q cms. To map the anterior boundary onto the standard life size diagram this distance of q cms is converted to life size value (Q cms) by multiplying it with the magnification factor. The anterior boundary of the lesion is then drawn on the diagram, and in figure 1a it is Q cms posterior to the reference axis (line AB). This procedure is repeated until the anterior and posterior lesion boundaries for all CT cuts are mapped onto the standard diagram.

### Lesion groups

Lesion groups were largely determined from the patient's CT scan. Lesions were classified (a) as anterior or posterior; (b) as right hemisphere or left

hemisphere; and (c) in terms of damage to particular subcortical sites. Lesions mapped onto the standard lateral diagram were often not completely confined to a particular site of interest, and in such cases several additional sources of information were used. First, in many cases an operation had been performed and part of the lesion was open to visual inspection. The investigator either attended the operation himself or he discussed the lesion site with the neurosurgeon who had performed the operation. Secondly, information about the neurological status for all patients at time of study was available in the notes, and could be used to resolve the issue. Thirdly, in some cases the investigator could not decide himself how to allocate a particular patient. When this happened a consultant neurosurgeon (Professor J.D. Miller), who was blind to emotional test results, reviewed the physical and neurological information and allocated the patient to one of the lesion groups.

**Anterior v posterior groups:** 15 patients had anterior lesions, 18 patients had posterior lesions, and in 13 cases the lesion could not be categorised as anterior or posterior. Anterior lesions were largely in front of the rolandic fissure. Lesions which extended a little beyond the rolandic fissure were included, if there was no evidence of somatosensory impairment or of a homonymous hemianopia. In the posterior group the lesion was largely behind the rolandic fissure, or within the temporal lobe. Patients whose lesions extended a little anteriorly to the rolandic fissure were also included if there was no evidence of a motor deficit.



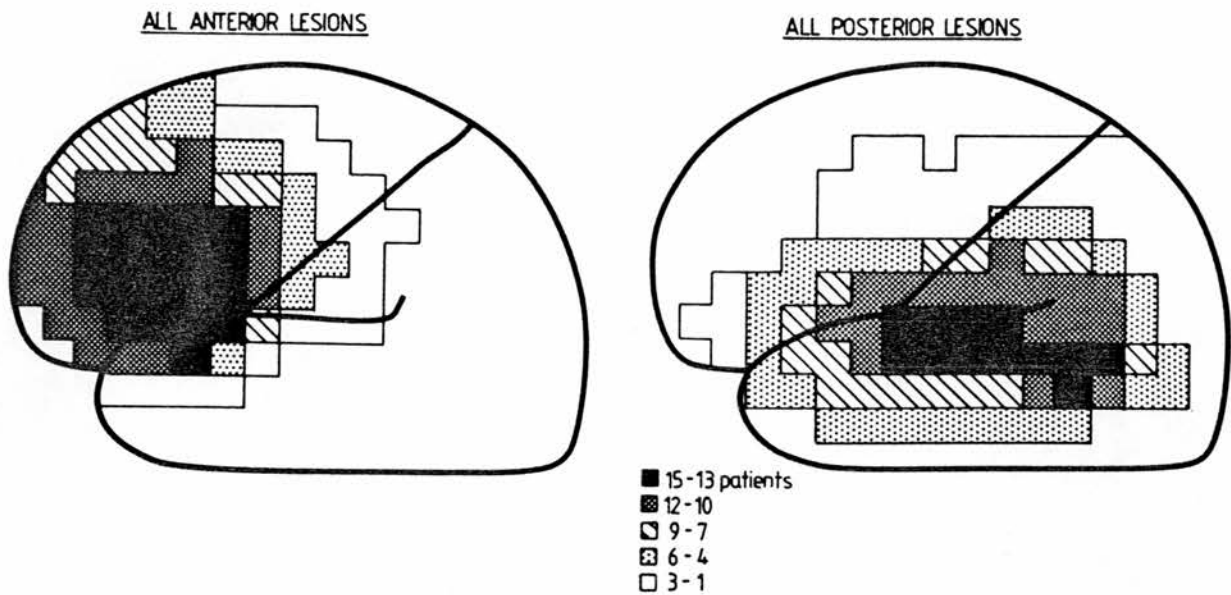


Figure 2

Composite lesion maps for patients with anterior and posterior lesions.

Composite lesion maps were constructed for the anterior and posterior groups (Figure 2). Individual patient lesion maps were all xerox reduced to the same size. A transparent grid of 1 cm squares was then placed over each lesion map. All squares which overlapped the lesion boundary were marked. This procedure was repeated for all patients in (a) the anterior lesion group and then, (b) the posterior lesion group. Figure 2 shows that the maximal damage in the frontal group is, in fact, anterior to the maximal region of damage in the posterior lesion group. However Figure 2 also shows that lesions are not evenly distributed in this sample. The anterior lesions are most commonly located in midfrontal and orbital regions, while the posterior lesions were most frequently situated in the perisylvian-superior temporal region. Furthermore,

there is significant overlap between the lesion groups. Many anterior lesions appear to encroach on the antero-medial temporal lobe. In fact, most of these lesions seemed to be deep frontal.], and medial temporal damage was noted in only 3 anterior patients. In the posterior lesion group there is also significant lesion extension beyond the rolandic fissure. This was due, in large part, to 6 patients. In two cases a metastatic lesion, represented by a high radiodensity region on CT, was located well behind the rolandic fissure. Though the associated oedema extended into the frontal lobe it was decided that these patients be included in the posterior group, given the evidence suggesting that such oedema might be associated with minimal functional deficit (Penn, 1980). The lesion maps of 4 other patients with meningioma (3 sphenoid wing and 1 parasagittal parietal) also suggested significant anterior extension. However following discussions with the operating neurosurgeon it was concluded that, in fact, there had been minimal anterior damage in these patients.

The anterior, posterior, and control groups were compared on demographic variables. The groups did not differ significantly for age [ $F(2,41)=0.33$ ;  $p=0.72$ ], sex distribution ( $X^2=0.16$ ;  $df=2$ ;  $p=0.92$ ), or social class (Kruskal Wallis oneway analysis of variance,  $X^2=0.49$ ;  $p=0.78$ ). However, from table 2 we can see that the anterior and posterior lesion groups were not matched for lesion type. All but one of the anterior group had a glioma, but less than half of the posterior group had this type of tumour. (Therefore when comparing the effects of anterior and posterior lesions it is important to control for the potentially confounding influence of lesion type.)

	<u>Anterior Group</u>	<u>Posterior Group</u>
Glioma		
(i) Astrocytoma(grade 1/2)	1	1
(ii) Astrocytoma(grade 3/4)	10	3
(iii) Oligodendroglioma	2	1
(iv) Unverified	1	1
Meningioma	1	4
Metastasis	0	3
Stroke	0	2
AVM	0	3

Table 2

Lesion aiology in anterior and posterior group

**Right hemisphere v left hemisphere groups:** 19 patients had unilateral right hemisphere lesions, 18 patients had unilateral left hemisphere damage, and lesions were bilateral in 9 cases. The right unilateral group consisted of patients whose CT scans showed a region of abnormal density largely confined to the right hemisphere. If a small abnormal density region was also noted in the left hemisphere the patient was still included in the right hemisphere group when neurological assessment found no evidence of dysphasia, or of right visual field defect, or right somatosensory impairment, or right motor impairment. Similarly patients were classified as having a left hemisphere lesion if the region of abnormal density was largely confined to the left hemisphere, and if there was no evidence of left sided visual, somatosensory or motor defects. The 9 patients who did not meet these criteria were classified as having bilateral lesions. The right and left hemispheric groups were roughly comparable in terms of lesion type (Table 3). Though the hemispheric and control groups did not differ for age [ $F(2,43)=0.12$ ;  $p=0.89$ ], or sex ( $X^2=0.17$ ;

$p=0.92$ ), patients in the right unilateral lesion group tended to come from lower social classes than controls or the left hemisphere patients (Kruskal Wallis  $\chi^2=5.2$ ;  $p=0.07$ ).

	<u>Right</u> <u>Hemisphere</u>	<u>Left</u> <u>Hemisphere</u>
Glioma		
(i) Astrocytoma(grade 1/2)	3	1
(ii) Astrocytoma(grade 3/4)	8	6
(iii) Oligodendroglioma	2	1
(iv) Unverified	1	2
Meningioma	1	3
Metastasis	3	0
Stroke	1	2
AVM	0	3

Table 3

Lesion aetiology in the right and left hemispheric groups

Given the evidence that anterior lesions and right hemisphere lesions may both produce emotional deficit, it was possible that especially marked emotional disturbance would be found with lesions, which are both anterior and in the right hemisphere. There were 3 right anterior, 4 left anterior, 9 right posterior and, 8 left posterior patients, and the composite lesion maps for these 4 groups are given in figure 3.

**Subcortical site:** Templates outlining the location of subcortical structures found at different CT cuts were made from the atlas of Hanaway *et al.* (1980). The templates were superimposed on the appropriate CT slice, and the lesion was said to invade the amygdala, hippocampus, thalamus, caudate nucleus, putamen, or globus pallidus if the total lesion boundary clearly overlapped with

these structures. Where mass effect caused significant shift an approximate adjustment was made, so that the lateral ventricles on the template partially overlapped with the lateral ventricles on the CT image.

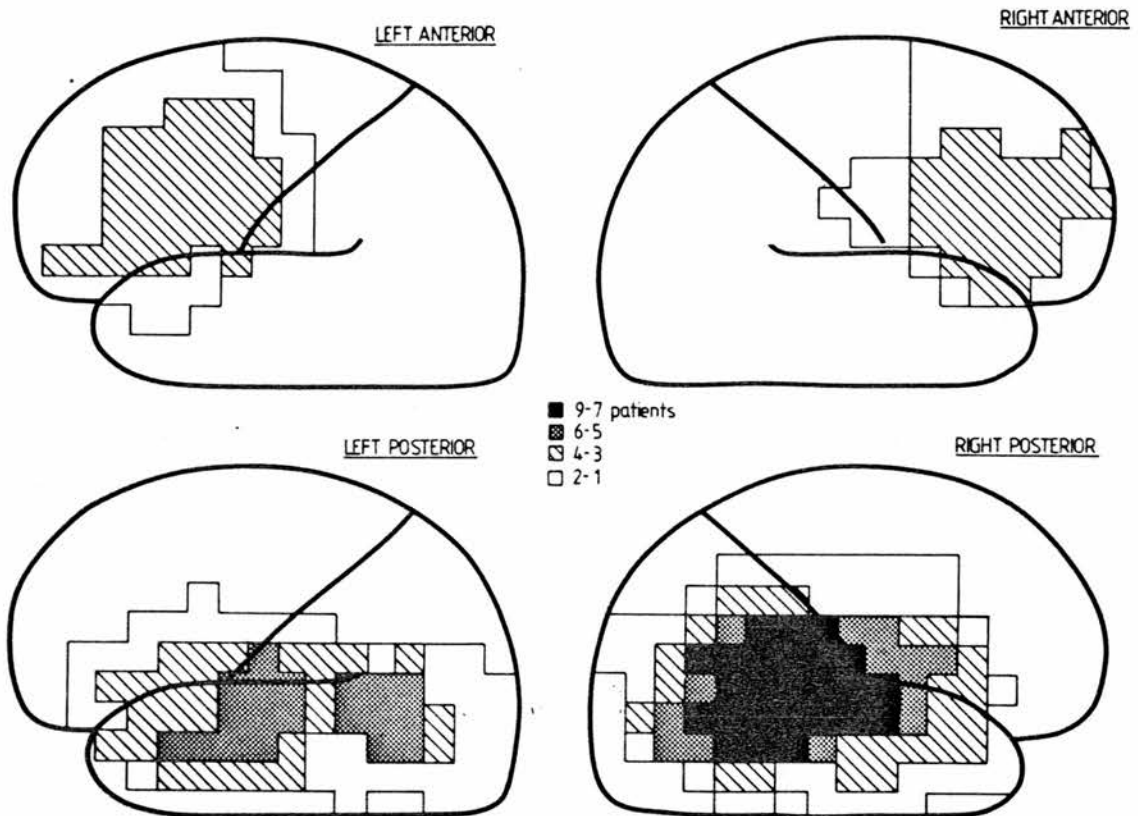


Figure 3

Composite lesion maps for right anterior, left anterior, right posterior, and left posterior patient groups.

Table 4 shows that 15 patients had no subcortical damage. In 13 cases the lesion boundaries encroached on one subcortical site. In the remaining group

12 subjects had damage to the basal ganglia and medial temporal structures; 6 had damage to the basal ganglia and the thalamus; 1 patient's lesion invaded both medial temporal and thalamic structures; and in 3 cases there was damage to basal ganglia, the medial temporal structures, and the thalamus.

<u>No sub- cortical damage</u>	<u>Basal ganglia only</u>	<u>Medial temporal only</u>	<u>Thalamic only</u>	<u>2 or more subcortical structures</u>
15	6	6	1	18

Table 4

Distribution of subcortical damage  
in patients with brain lesions

### **3.4. Assessment of Cognitive, Perceptual and Speech Functions**

#### **Cognitive testing**

**WAIS:** To assess intellectual status while keeping the amount of testing within limits tolerable to the patient, three verbal and three performance subtests of the WAIS were administered. The subtests were Digit Span, Similarities, Arithmetic, Picture Completion, Block Design and Picture Arrangement. A prorated estimate of verbal and performance IQ was gained from the average of the scaled scores of the verbal and performance subtests.

This shortened version of the WAIS is the same as that used in the Psychology Department of the National Hospital, Queens Square, London (Warrington *et al.*, 1986), except that the Vocabulary subtest was excluded because some patients take a long time to complete this subtest.

**Modified Wisconsin Card Sorting Test:** With one exception (Teuber *et al*, 1951) several workers have found that patients with frontal lobe damage make significantly more perseverative errors on the Wisconsin Card Sorting Test (Robinson *et al*, 1980; Nelson, 1976; Drewe, 1974; Milner, 1963). The patient was required to consecutively sort individual response cards under one of the following 4 key cards; 1 red triangle, 2 green stars, 3 yellow crosses, and 4 blue circles. Each response card contained a certain number (1,2,3 or 4) of shapes (triangle, star, cross or circle) of a particular colour (red , green, yellow or blue). The patient was to sort each response card under one of the key cards according to one of the three possible rules; the patient could sort by shape, colour or number. The response cards recommended by Nelson (1976) were used. These response cards share one and only one attribute with each of three key cards (e.g. the same colour as the first, the same shape as the second and the same number as the third) and shared no attribute with the fourth card. Thus if the patient places the response card of one green cross under the key card with three yellow crosses the experimenter knows the patient is sorting according to shape. While if sorting according to colour, this one green cross would have been placed under the key card with two green stars. If the patient had been sorting by number the one green cross would have gone under the one red triangle. After 6 consecutive correct responses, without warning the rule was changed and the patient had to work out the new rule.

This procedure was generally followed for at least 20 card sortings. Subsequently procedural changes were made for reasons to be outlined below. However these initial 20 card sortings gave some indication of perseverative tendency i.e. the tendency to persistently follow a sorting rule in spite of being quite clearly told that this rule is incorrect. The patient scored one



perseverative error if his card sort followed the same category concept as an immediately preceding incorrect response. The ratio of number of perseverative errors divided by total errors was calculated as this measure has been found to discriminate frontal from non-frontal groups (Nelson, 1976).

**Recognition Memory for Faces and Words:** To briefly assess memory a modified version of Warrington's (1974) test of recognition memory was used. The stimuli consisted of 24 high frequency (Thorndike and Lorge; 1944) 4 to 5 letter words and 24 full face photographs of unknown actors. Single faces and words were presented alternately, each for 3 to 4 seconds. Stimulus orientation has been shown to facilitate recognition and so the subject was required to say whether each stimulus was "pleasant" or "unpleasant". The subject was then presented with alternate pairs of words or pairs of full face photographs. Though the patient had previously seen only one member of each pair he was not informed of this. He had to decide whether he recognised neither, both or only one of the pair of words or faces. The patient scored one point only when he identified the correct item of the pair. On this test a patient could score a maximum of 24 points for facial recognition and 24 points for word recognition.

**Benton Visual Retention Test:** The previous recognition memory test was designed in the hope that it would be able to detect material-specific memory deficits. There is no good single test of memory function and though the Benton Visual Retention Test is not a pure test of memory (Russell, 1981), it is well standardised.

The patient was presented with a series of 10 increasingly complex designs. After each design has been presented for 10 seconds the patient must draw it from memory. Thus patients can score 0 to 10 points on this test. The ability

to remember designs lessens with increasing age and so, in the present study the age corrected score was used.

### Perceptual testing

**Recognition of facial identity:** The patient had first to identify 9 full face test photographs. Each test photograph was presented singly and on its removal the patient had to pick out the identical photograph from an array of 6 response photographs. There were three arrays of 6 response photographs. Each array was composed of 3 test photographs and 3 distractor photographs which the patient had not seen before. Thus in all the patient could correctly identify a maximum of 9 faces after a delay of 0 seconds.

The patient had to identify a further 9 individuals according to an almost identical procedure. The only procedural change was that after seeing the test photograph the patient had to wait 4 seconds before he had the opportunity of identifying it in the array of 6 photographs. Thus the patient could identify a maximum of 9 faces after a delay of 4 seconds.

**Visual Neglect:** Two simple quantitative measures of left sided visual neglect were administered only to patients with right hemisphere damage. In a cancellation task (Damasio *et al.* 1980) the patient was shown a sheet of A4 paper with ninety 2 cm. lines of all orientations evenly distributed over a central area of 17x15 cms. E crossed out two lines in the centre of the array whose midline was in line with patient's own vertical midline, and the patient was told to cross out all of the remaining eighty-eight lines as quickly as possible, and to say when he had finished. The patient scored one error for every line missed to the right or left of the midline. The index of neglect derived was number of lines missed on the left.

In a line bisection task (Schenkenberg *et al.*, 1980) eighteen lines were drawn on a sheet of A4. The lines were organized in three sets of six lines so that one set of lines lay primarily on the left side of the page, one set lay in the centre, and one lay on the right. Each set contained lines of 100mm, 120mm, 140mm, 160mm, 180mm, and 200mm. The patient was asked to cut each line in half by placing a small pencil mark through its centre. Percent of right deviation was measured for each of the left sided set of lines according to the equation:  $\text{Percent deviation} = 100 \times (\text{measured left half} - \text{true half}) / (\text{true half})$ . The patient's score was the average percent of right deviation on the 6 left sided lines.

### Dysphasia screening

A short form of the Token Test was administered only to patients with left hemisphere damage. This index of verbal comprehension was used as a screening device to identify those patients with dysphasia. It is a well established test consists of 20 differently coloured square and round tokens of two sizes. The patient is given increasingly complicated instructions requiring him to touch or move the tokens. Normative data is provided by De Renzi and Faglioni (1978), and a cut-off score of 29/30 was used to distinguish dysphasic from non-dysphasic subjects. The Token Test also gives some indication of severity of dysphasia.

Gainotti (1972) suggested that there were differences in the emotional reactions of fluent and non-fluent dysphasics. However, piloting indicated that it would not be possible to recruit enough patients to meaningfully investigate the effects of type of dysphasia. Since the test battery was large, and patients with non-fluent dysphasia can be very frustrated by their word finding difficulties, oral expression was not routinely quantitatively tested. Of course,

the investigator always made a clinical assessment of the degree of anomia, paraphasia or paragrammatism in free speech, followed by more systematic testing where dysphasia was suspected. In fact, all patients below the cut-off score on the Token Test had significant difficulties with verbal expression. Conversely, patients scoring above the Token Test cut-off generally had minimal or no obvious disorder of verbal expression. In one case, however, the Token Test score was 30 but the patient had moderately severe word finding difficulties. This patient was therefore classified as dysphasic.

### **3.5. Tests of Perception of Emotion**

#### **Test of perception of facially expressed emotion**

The test stimuli were two groups of 20 full face photographs selected from the Ekman and Friesen (1976) series. The faces express one of seven feeling states, and Ekman and Friesen have shown that there is a high degree of consensus about the emotion expressed in each photograph. Both groups of 20 included 3 happy, 3 sad, 3 angry, 3 surprised, 3 fearful, 3 disgusted and 2 neutral faces (See Appendix 1). Faces were presented singly for 2 to 3 seconds. After a delay of 0 seconds, for the first 10 photographs, and of 4 seconds for the second 10 photographs the patient was required to match every test face presented with one of a set of standard faces each expressing one of the seven emotions. At a later stage the subject was presented with the second group of 20 faces. As before after a delay of either 0 or 4 seconds the subject was asked to match each face with one of seven verbal labels; happy, sad, angry, fearful, surprised, disgusted or neutral. Several measures were derived from this test.

- The total number of emotional faces correctly identified was scored.

- A separate count was made of the number of faces identified when matching to (a) verbal labels and (b) to standard faces.
- A record was made of the number of faces of each of the 7 emotional types correctly identified.
- Each patient had a score for the number of faces identified after a delay of 0 seconds and after a 4 second delay.

### Test of perception of emotion expressed in speech and text

The test stimuli used were 44 short emotional utterances recorded from radio plays, together with 28 emotional sentences presented in written form. Clearly in the written sentences the emotion could only be expressed purely in the verbal-semantic content. However each tape-recorded utterance had to be classified as either expressing emotion purely in the tone of voice or as expressing emotion in both tone and in verbal-semantic content. This classification was achieved by subjecting every tape-recorded utterance to three separate groups of 10 non-brain injured raters, who were all hospital staff volunteers.

First, the tape recorded utterances were presented to a group of 10 subjects who had to listen to the utterance as a whole and decide whether the speaker was happy, sad, angry, neutral or surprised. Sentences were included in the test only if a minimum of 6 raters agreed on the emotional significance of the utterance. In fact agreement was generally higher than the minimum level, and an average of 8.5 raters agreed about the emotional significance of each sentence. Secondly, the tape recorded utterances were played to a different group of 10 subjects who had to ignore the verbal-semantic content of the sentences. They had to decide whether the tone of voice was happy, sad, angry, neutral, or surprised. Though agreement between at least 6 raters was necessary before an utterance could be included in the test, an average of 9.2

raters agreed about the emotion expressed in the tone of each sentence. Thus the utterances which remained after having been subjected to these two ratings were known to express a particular emotion in the tone of voice. It only remained to be discovered whether the emotion was **also** expressed in the verbal-semantic content of the utterance. To decide this, written transcriptions of the tape-recorded utterances were categorised as happy, sad, angry, surprised or neutral by a final group of 10 judges. As the sentences were presented in written form these judges were forced to rely solely on the verbal-semantic content to make their assessment of emotional significance. This third group of raters made it possible to dichotomise the remaining sentences as expressing emotion either **solely** in tone of voice or as expressing emotion **both** in tone and in content. Thus a sentence was classified as expressing a particular emotion (e.g. happiness) only in the tone of voice if no more than 2 raters (mean = 0.3 raters) who assessed the verbal content judged that sentence to express this particular emotion (e.g. happiness). Conversely, emotion was said to be expressed in both tone of voice and in content if at least 6 raters (mean = 8.4 raters) judged the sentence to be happy. Finally, in order to produce stimuli where emotion was expressed purely in the verbal-semantic content of the sentence this third group of raters were also asked to assess the emotional significance of a further corpus of written sentences.

This procedure generated 3 types of sentence (See Appendix 1). There were 22 spoken sentences which were categorised as expressing emotion purely in tone of voice (these comprised 8 happy sentences, 7 angry sentences and 7 sad sentences). There were a further 22 spoken sentences where emotion was said to be expressed in tone and content (8 angry sentences, 6 sad sentences, 4 happy sentences and 4 neutral sentences). Finally there were

28 written test sentences where emotion was said to be expressed in the verbal-semantic content (8 happy, 8 angry, 5 sad and 7 neutral sentences). In short, in the test of perception of emotion expressed in speech and text the **stimuli** were systematically varied i.e. specific, and different, types of emotion were expressed in tone and/or content.

In addition to varying the stimulus characteristics, the **response** requirements were also systematically manipulated. For this the tape-recorded and written test stimuli were split into two roughly comparable groups. The split was carried out in the following manner. The 28 written sentences were subdivided into two groups of 14 sentences, each roughly comparable in terms of difficulty and emotional type. The 44 tape recorded sentences were also subdivided into two groups roughly matched for difficulty and emotional type. However in addition, for each group of 22 tape-recorded utterances, the initial 11 utterances had emotion expressed in both tone and content. For the next 11 utterances the patient was informed that emotion was expressed solely in tone of voice. Response requirements were manipulated using these comparable groups of emotional stimuli. Patients had to match the first half of the test stimuli to faces, and the other half to words. For the first half of the test stimuli the patient had to identify the emotion expressed by pointing to a happy, sad, angry, neutral or surprised face selected from the Ekman and Friesen (1976) series. In the second half of the test the subject had to identify the emotion by pointing to one of 5 verbal labels; happy, sad, angry, neutral or surprised. (Though there were no surprised test sentences the surprised category was included to increase test difficulty.) Several measures were obtained from this test.

- A record was made of the total number of utterances where the emotion was correctly identified.



- A note was made of the number of correctly identified utterances where emotion was expressed in tone; or in tone and content; or solely in content.
- The number of utterances identified when matching to standard faces, and when matching to verbal labels.
- A count was taken of the happy, sad, angry and neutral sentences that had been correctly identified.

### **3.6. Assessment of Emotional Expression and Facial Movements**

The emotional responses of neurological patients have only occasionally been filmed (Strauss *et al*, 1983; Buck and Duffy, 1980). This is unfortunate because emotional expressions can clearly be studied with greater precision when a permanent record of the patient's emotional responses is available. In the present study the patient's facial, head and vocal responses were videotaped as he participated in standard experimental tasks designed to elicit affective and non-affective behaviours. The experimental set-up is illustrated in figure 4, which shows the patient facing a video camera which was behind a glass window 4 feet away from him. To obtain high definition recordings of facial expression, only the face and head were filmed. The experimenter stood opposite the patient. His head was at the same level as the camera lens and only a few inches to the left of it. This strategy made the video equipment unobtrusive while at the same time it ensured that the patient's face was constantly directed at the camera lens throughout the administration of the standard tasks.

#### **Spontaneous Reaction to Failure**

Facial reactions were filmed as the patient performed the above described Wisconsin Card Sorting Test. Briefly, the patient had to find the correct way of sorting response cards under 4 key cards. He had to work out the correct

sorting principle by trying out different rules and after each sorting attempt the experimenter told him that he was right or that he was wrong. In order to ensure that the facial expressions elicited were spontaneous the patient was not informed that we were filming his reactions to card sorting. Instead he was told that we were filming the postural changes that occur during the performance of a concentration test.

The Wisconsin Card Sorting Test is not equally difficult for all patients. Therefore one lesion group may show a greater spontaneous emotional reaction in this situation merely because he has greater difficulty with this task. Conversely, another lesion group may show fewer reactions because, for them, this task is easy. In other words, reactions elicited may be more related to task difficulty than to ability to regulate emotional responses. To minimise the confounding influence of task difficulty all patients sorted 40 cards and 20 of these were said to have been incorrectly sorted. Thus, following an initial phase when a measure of perseverative tendency was derived, procedural changes were introduced. Correct and incorrect trials were fairly evenly distributed throughout the task because the patient was never correct (or incorrect) for more than 12 consecutive trials. To ensure even distribution of failure and success the task was sometimes made impossible to solve, and on other occasions strong hints about the correct solution were given. Finally the last 6 trials were always judged incorrect. This attempt to equalise test difficulty appeared to be reasonably successful. Certainly most patients expressed surprise when they were informed that the experimenter had arranged matters, with all patients making precisely 20 incorrect card sortings. Qualitative, quantitative, self-report, and autonomic indices were used to assess the patient's emotional responses to this situation.

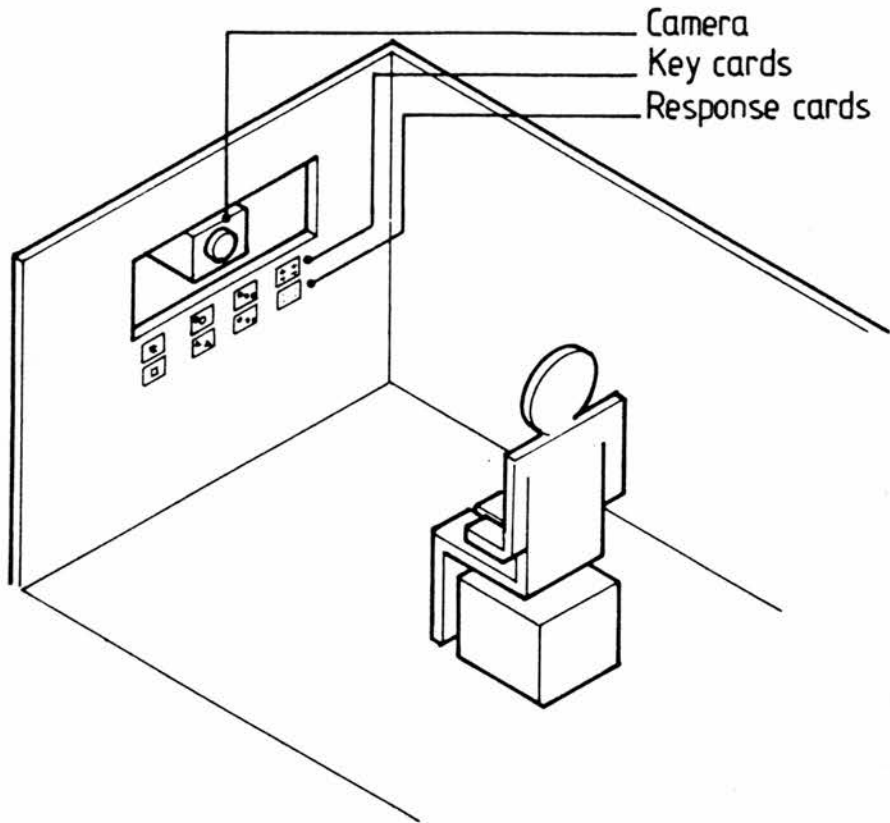


Figure 4

During assessment of facial movement and expression, the patient sits facing a video camera lens, and just below this is a small shelf on which are placed the response cards of the Wisconsin Card Sorting Test.

**Quantitative score:** The patient's facial expression was rated over a time period bounded by two remarks made by the experimenter. Each period began as soon as the experimenter had finished saying "wrong", and ended at the start of the next trial when the the experimenter asked "Now where does this next card go?". The first 10 reactions after having been told he was wrong were rated using the well researched Facial Action Coding System (FACS) of Ekman and Friesen (1978). FACS defines facial movements in terms of 44 reliably

measurable Action Units (See Appendix 1). For example, Action Unit 12 consists in the contraction of the Zygomatic Major muscle which pulls up the lip corners and raises up the lower-middle portion of the nasolabial furrow. Each facial expression was scored for the total number of action units produced. The experimenter and another judge independently assessed number of action units in 15 facial expressions, and a correlation of 0.92 was obtained. In addition to counting total number of action units, it was thought possible to fairly reliably count number of head movements, comments, and vocalisations for each reaction.

**Qualitative score:** Ratings of the first 10 responses to being told he was incorrect were also made. Each of the patient's reactions was rated on a 4 point scale for the presence of negative affect. The reaction scored 2 points if negative affect was clearly present, and 3 points if the negative affect was strong or marked. 0 points were given when there was definitely no negative feeling or if negative feeling was just a **possibility**. However when negative feeling was **very probably** present a score of 1 point was awarded. The ratings for the 10 reactions were summed to give a measure of the degree of negative reaction to failure. These same 10 reactions were then reassessed for the presence of positive affect using an identical 4 point scale. Qualitative ratings were also made of the first 10 reactions to being told he was correct. The same 4 point scale was used for both positive and negative reactions to being correct. The experimenter and another judge independently rated 10 subjects and obtained correlations of 0.88 for negative reaction, and 0.85 for positive reaction after an incorrect card sort. While after a correct card sort the interjudge correlation was 0.88 for positive reactions. It seemed to one judge that occasionally patients had a negative reaction after a correct card sort, but one judge never rated negative response on these occasions, and so the

interjudge reliability for negative reaction during a correct card sort was not computable.

**Self-report** After card sorting was completed the patient was told that we wished to check that the card sorting test was not unduly distressing. This was done to establish the degree of association between the patient's subjective emotional experience and his capacity to express affect. The patient was asked to rate the task on 4 positive adjectives (interesting, enjoyable, fun to do and satisfying) and 6 negative adjectives (anxiety provoking, confusing, frustrating, irritating, childish and upsetting). If he agreed with the adjective "Yes, very much so" he scored 3 points; "Yes, a little" earned 2 points; "No, not much" gained 1 point; and "No, not at all" scored 0 points. This gave a maximum positive score of 12 and a maximum negative score of 18.

**Autonomic response:** The patient's heart rate was used as an index of the autonomic component of the patient's emotional reactions. Piloting showed that there was very little movement artifact on the heart-rate trace, when the 3 electrodes were placed on the patient's waist. Electrodes were positioned as follows: 2 inches above the iliac crest of the right pelvis, 2 inches above the iliac crest of the left pelvis, and the earth electrode was placed above the navel. Baseline heart-rate was measured at the beginning of the filmed session when experimenter and patient were discussing neutral topics. The mean heart rate during the first 10 incorrect, and the first 10 correct, sortings was also recorded.

### The voluntary production of emotional expressions

These were considered because it has been argued that **voluntary** and **spontaneous** facial expressions are controlled by different neural systems

(Ekman *et al.*, 1981; Raleigh and Steklis, 1979). The patient was first shown one of the Ekman and Friesen (1976) photographs of a person smiling. The experimenter said "This person is happy. Something very good has happened to him. He's smiling because he is so happy. I want you to imagine that, like him, you are happy and smiling – so smile." Appropriate versions of this procedure were subsequently followed when the patient was asked to produce disgusted, surprised and, finally, angry facial expressions.

The film sequence which was judged by the experimenter to be the patient's best attempt was rated using FACS to provide a quantitative measure of the **amount** of facial movement produced. However patients sometimes made considerable facial movements and yet were clearly unsuccessful in their attempts to portray a particular emotion. It was therefore also desirable to assess the adequacy of each of the patient's expressions. Such qualitative ratings were made on a 4 point scale. The expression scored at least 2 points if the patient's expression was clearly adequate. If the emotion expressed was judged to be adequate and very strong, it scored 3 points. On the other hand, the patient's expression scored 0 points if it was totally inappropriate and bore no relation to the emotion requested. If, however, the expression did appear to contain some of the elements of the requested emotion a score of 1 point was awarded. As there were 4 facial emotional expressions the patient could get a maximum qualitative score of 12 points. With 4 months between ratings, test-retest reliability for number of FACS action units was 0.86; and for total qualitative score was 0.95.

### Non-emotional facial movements

**Measurement of upper face movements during speech:** As we have seen impairment on the above emotional tasks may not always be attributable to a

primary emotional deficit. Impairment on the emotional tasks may be due to a more general motor deficit. To assess non-emotional facial movement a frequency count was made of upper face movement during speech. The experimenter asked the patient to give a history of their symptoms. By this means it was generally possible to produce 30 seconds of film where the patient was talking in a neutral, non-emotional fashion. For most patients the 30 seconds consisted of 1 or 2 continuous film sequences. However for a few patients, up to 4 separate sequences were required. The 30 second film was rated for the presence of eye brow and eye movements (FACS action units 1,2,4,5,6, and 7). Using 15 subjects the test-retest correlation, with 4 months between ratings, for total upper face movement was 0.82.

**The imitation of meaningless facial movements:** This was a modified version of a test used by Kolb and Milner (1981a). The patient was first asked to imitate three facial movements involving the lower face; bilateral lip retraction; showing the tongue with mouth otherwise closed; and bite lower lip. He was given up to 3 attempts to imitate each single movement. The experimenter then performed the three mouth movements in sequence and the patient was required to copy this. If the patient made any errors he was shown the correct sequence up to twice more. The patient had then to imitate three upper face actions, singly and in sequence; close both eyes, brow lower (as in frowning), and wink the right eye. The final group of 3 movements involved both the upper and the lower face. The patient had to raise his eyebrows, open his mouth wide, and close both eyes.

There were three types of error scored; (a) an omission error was scored when a movement element was not recalled; (b) addition errors occurred when an incorrect movement was substituted; (c) a position error was scored if a correct movement was produced in the wrong position. For example, if the



patient produced the second movement first and the first movement second, 2 position errors were scored, and clearly no addition or omission errors would be recorded. Errors of all three types were summed to give the total facial imitation error score.

It was expected that this task would detect the presence of oral apraxia because patients have to imitate lower face movement sequences. This test may be sensitive to other facial movement disorders, because upper facial movements are required.

### **3.7. Administration of Tests and Statistical Analyses**

Patients were tested early on in recovery, as soon as they felt able to tackle the test situation. Generally testing was completed in 4 sessions. In the first session all patients were administered the WAIS and the Benton Visual Retention Test. Those with left hemisphere damage were also administered the Token Test, while right hemisphere patients were asked to complete the line bisection and the cancellation tasks. The Token Test was only administered to left hemisphere patients, to keep testing time within reasonable limits. This was felt legitimate because dysphasia is usually due to left hemisphere damage in right and left handers (Hecaen and Sauguet, 1971). Furthermore, only one left handed patient had a right hemisphere lesion and, certainly, the investigator's clinical impression was that none of the present right hemisphere patients was dysphasic. Similarly, the line cancellation and bisection tests were administered only to patients with right hemisphere lesions, because left unilateral neglect is more common than right neglect (Gainotti, 1968) and, indeed, none of the present patients with left hemisphere lesions displayed clinically obvious signs of neglect.

At the end of the first session the patient was told that we were trying to develop tests of vision, hearing and movement. This work was at an early stage and we simply required patients to give us their opinion about the value of these different tests.

In the second and third sessions the tests of perception of facially and verbally expressed affect were administered. The response requirements for these tests were systematically manipulated. In the second session the patient was required to match half of the emotional test items to standard facial response cards (expressing happiness, sadness, etc). It was hoped that when matching to standard emotional faces the patient would rely less on language than in the third session, where the remaining emotional test items were matched to standard emotional words (happy, sad, etc). The recognition memory and the recognition of facial identity tests were also administered at the end of the second and third sessions respectively. The remaining tests of expressive functions were completed in the final session.

All statistics were computed using SPSSX (SPSSX Inc., 1983). Emotional test performances of all patients with anterior lesions were first compared with all patients who had posterior damage. In the second phase, emotional test performance in the anterior and posterior groups were compared after the influences of lesion variables (lesion size, and type), and general sensori-motor-cognitive variables (IQ, memory, and perceptual deficits) were statistically controlled. A similar procedure was followed for the comparison of emotional test performances of the right and left hemisphere groups.

The majority of patients in this study were seriously ill and, consequently, it was not always possible for them to complete the whole test battery(See Appendix 2). Because of this missing data the numbers of cases in the

statistical analyses is variable. The number of subjects available for any particular analysis is quoted in the text, when group size is reduced because of missing data and/or the introduction of statistical controls.

## CHAPTER 4

### THE ROLE OF THE FRONTAL LOBES IN THE PERCEPTION OF EMOTION

The emotional perceptions of patients with anterior and posterior lesions were compared on two tests. In the first test the patient was shown pictures of faces expressing emotion and he had to identify the emotion expressed. In the second test the patient had to determine the affective quality of emotion conveyed in speech and text. If the frontal lobes are specialised for the regulation of all emotional processes it would be predicted that patients with frontal lesions would have more difficulty than patients with posterior lesions on both tests of perception of emotion.

#### 4.1. Test of Perception of Facially Expressed Emotion

Briefly, the patient was shown happy, sad, angry, surprised, fearful and neutral full face photographs selected from the Ekman and Friesen (1976) series. After a delay of 0 seconds, or of 4 seconds, the patient was required to identify the feeling expressed. This was done either by (a) matching each test face with one of a set of 7 standard emotional faces or (b) matching each test face with the corresponding 7 emotional verbal labels; happy, sad, angry, fearful, surprised, disgusted, or neutral. Several measures were derived from this test:

- Total emotional faces correctly identified.
- The number of faces identified when matching to (a) verbal labels and (b) to standard faces.
- The number of faces of each of the 7 emotional types correctly identified.
- Total faces identified after (a) a delay of 0 seconds and after (b) a 4 second delay.

In a 3x2 analysis of variance total scores on the perception of facially expressed emotion was the dependent variable, and lesion site (control v anterior v posterior), and sex (male v female) were factors. Only lesion site had a significant influence on test scores [ $F(2,34)=22.84$ ;  $p<0.0001$ ]. Neither sex [ $F(1,34)=0.78$ ;  $p=0.38$ ], nor the sex by site interaction term [ $F(2,34)=0.45$ ;  $p=0.64$ ] achieved significance. Therefore in subsequent analyses the 21 male and the 19 female subjects are grouped together.

<u>Score Range</u>	<u>Anterior Group</u>	<u>Posterior Group</u>	<u>Control Group</u>
40-37			1
36-33		1	5
32-29	1	2	4
28-25		4	
24-21	2	6	
20-17	3	1	
16-13	4	2	
12-9	1	1	
8-5	1		
4-0	1		

Table 5

Total scores on the test of facially expressed emotion for the control, anterior and posterior groups. The number of subjects scoring within a particular range is represented e.g. 1 control subject, but no anterior or posterior subjects, scored within the range of 40 to 37 points.

Total scores for the 10 controls, the 13 anterior and the 17 posterior lesion groups are given in Table 5, and in a one-way analysis of variance the group effect was significant [ $F(2,37)=23.29$ ;  $p<0.0001$ ]. Post hoc Scheffe testing showed that the control group was significantly superior to patients with posterior, and with anterior lesions. This would indicate that the anterior and

posterior brain normally **both** contribute to the efficient perception of facially expressed emotion. However post hoc testing also showed that the anterior group had significantly lower scores than the posterior group. The particularly marked impairment in the anterior group is consistent with the hypothesis that the frontal lobes are specialised for perception of emotion; but before accepting this latter hypothesis we must eliminate several alternative explanations for the greater frontal deficit.

### Perception of facially expressed emotion with lesion size and location

**Lesion size and aetiology:** Though patients with anterior lesions generally performed less well than patients with posterior lesions on the test of perception of facially expressed emotion, average lesion size tended to be larger in the anterior group (108.6cc; SD=45.4cc) than in the posterior group (86.6cc; SD=47.3cc). While not statistically significant ( $t=1.36$ ;  $df=31$   $p=0.19$ ), this measure of difference in lesion size between groups is likely to be an underestimate. Lesion size was determined from pre-operative scans and at operation frontal excisions tend to be larger than posterior excisions. This increases the possibility that the uncontrolled influence of lesion size accounts for the greater frontal impairment. To exert some control over the confounding influence of lesion size, all patients with frontal lobectomies were excluded from the comparison between anterior and posterior groups. Only the 8 frontal patients who had no more than a decompression and tumour debulking were contrasted with the 17 patients who had posterior lesions (two of whom had had one occipital lobe removed). In a one-way analysis of covariance total lesion volume was introduced as a covariate. Lesion volume did tend to influence facial emotion test scores [ $F(1,21)=4.23$ ;  $p=0.06$ ], but after this effect

was partialled out perception of facially expressed emotion was still significantly more impaired in the anterior group [ $F(1,21)=12.46$ ;  $p=0.002$ ].

A further potential confounding influence stems from the anterior and posterior groups being unmatched for lesion aetiology (Table 2). All but one patient in the group of patients with anterior lesions had a glioma, whereas two posterior patients had a metastasis, three had had an AVM excised, one had had a stroke, and meningiomas were more common in the posterior group. Since lesion aetiology may influence severity (Warrington *et al.*, 1986) and type (Reitan, 1964) of cognitive deficit, the anterior-posterior difference in emotional perception may also merely reflect group differences in lesion aetiology. Therefore anterior and posterior groups matched for lesion type were compared. The anterior group comprised 7 glioma patients together with one patient who had a meningioma, while the posterior group consisted of 5 glioma cases and 1 of the meningioma patients. All the patients with frontal lobectomy, a metastasis, AVM, and stroke were excluded. The anterior deficit remained, after making adjustments for pre-operative lesion size [ $F(1,11)=4.91$ ;  $p=0.047$ ].

Another difficulty is that, though one posterior patient had bilateral damage, 6 anterior patients had some bilateral involvement. While we know of hemispheric functional asymmetries, there is also good reason to believe that homologous regions in both hemispheres can sometimes perform similar functions. For this reason bilateral damage is liable to produce more severe deficit than unilateral damage, and certainly this was what Benton (1968) found. Furthermore, the anterior patients were subdivided into those with ( $N=6$ ) and those without ( $N=7$ ) bilateral damage, and significantly greater deficit on the test of perception of facially expressed emotion was found in the bilateral subgroup (Mann-Whitney  $U=6.5$ ;  $p=0.035$ ). Therefore the anterior and posterior



groups were matched for the degree of bilateral involvement. The anterior group comprised 7 patients with unilateral frontal damage and one patient with bilateral damage. The posterior group consisted of 16 patients with unilateral posterior damage and 1 patient with bilateral involvement. Only a non-significant trend for greater anterior deficit remained after this control ( $t=1.58$ ;  $p=0.13$ ).

**Lesion location:** For this study we have contrasted anterior and posterior groups, but these are large neural regions. Anatomical, electrophysiological and lesion evidence all show that distinguishable neural systems are contained within these regions. For the frontal lobes there have been attempts to differentiate the emotional sequelae of dorsolateral from ventral/orbital frontal damage. For the posterior group it is desirable to contrast anterior temporal lesions v posterior temporo-occipital lesions v parietal lesions. However in the present study we were unable to recruit a sufficient number of suitable cases for a full analysis of effects of lesion locus within a cerebral quadrant, because patients' lesions tend to occur most frequently in mid and basal frontal, as well as perisylvian sites (See Figure 2).

Extent of damage to subcortical structures may also influence perception of facial emotion scores. To examine this patients with damage to 1 or more subcortical structures ( $N=29$ ) were compared with patients who had no subcortical damage ( $N=14$ ), but no significant difference was found for perception of facial emotion ( $t=0.89$ ;  $df=41$ ;  $p=0.38$ ). While the presence of subcortical damage *per se* did not significantly increase the likelihood of greater deficit on the facial emotion test, it remains possible that particular subcortical structures (such as the amygdala) are especially involved in the perception of emotion. To consider this, the 17 patients with damage to the amygdala and/or the hippocampus were compared with the 27 patients who

had no damage to these structures. Perception of facial emotion scores were similar for both groups ( $t=0.08$ ;  $df=42$ ;  $p=0.94$ ). When patients with damage to the any part of the basal ganglia ( $N=21$ ) were compared with all other patients ( $N=23$ ) a significant difference was found ( $t=2.41$ ;  $df=42$ ;  $p=0.021$ ). However lesion size was larger in the patients with damage to one of the structures of the basal ganglia ( $t=2.32$ ;  $df=43$ ;  $p=0.025$ ). To control the joint influence of lesion size and depth, only those patients with damage to one subcortical structure were considered in a subsequent analysis. 3 patients with damage to only the basal ganglia were contrasted with the 6 patients whose damage extended into either the medial temporal ( $N=5$ ) or into thalamic ( $N=1$ ) structures. However in this comparison the group with damage to the basal ganglia no longer showed significantly greater impairment (Mann-Whitney  $U=4$ ,  $p=0.2$ ).

#### Perception of facial emotion, lesion site, and cognitive function

The greater impairment of the anterior group on the test of perception of facially expressed emotion suggests a defective emotional function in this group. However a selective impairment of specifically emotional perceptions is only one of the potential causes of failure. Impaired performance may result from a deficit in memory or other perceptual/cognitive function. One way of addressing this issue is to manipulate the stimulus and response requirements of the test.

In an attempt to examine perceptuo-cognitive factors, patients were required to match stimulus faces to standard emotional faces, or to standard emotional names. Furthermore, matching was also done immediately, or after a 4 second delay. Under all conditions the anterior group remained significantly more impaired (Table 6). Using the SPSSX multivariate repeated measures

analysis of variance procedure, significantly more errors were produced with the longer 4 second delay [ $F(1,28)=7.892$ ;  $p=0.009$ ], but there was no significant difference in performance between matching to faces and matching to words [ $F(1,28)=1.59$ ;  $p=0.2$ ]. For all manipulations of stimulus and response requirements, the anterior lesion group was impaired relative to the posteriors, and the interactions of site by stimulus condition (0 secs v 4 secs delay), and of site by response requirements (face v word matching) both fell short of significance; with F-values of 0.05 ( $df=1,28$ ), and 1.76 ( $df=1,28$ ), respectively.

	<u>Anterior Group</u>	<u>Posterior Group</u>	<u>t-value</u>	<u>p value</u>
<b>Total Score</b>	16.3	23.2	2.98	0.006
<b>Response</b>				
Face match	7.7	11.2	2.64	0.013
Word match	8.6	11.9	2.60	0.015
<b>Delay</b>				
0 seconds	9.3	12.1	2.22	0.044
4 seconds	7.0	11.2	2.99	0.006

Table 6

Mean scores for the anterior and posterior groups on some measures derived from the test of perception of facially expressed emotion.

There is an alternative strategy for distinguishing emotional from general perceptual-cognitive explanations for the anterior deficit on the test of facially expressed emotion. In addition to tests of emotional perception, measures of general perceptual-cognitive function were also administered. The facial identity recognition task corresponded to the test of perception of facially expressed emotion in several important respects, but no perception of emotion

was involved. The patient was shown a test face and required to point to the identical photograph from six alternative response photographs after a 0, or 4 second, delay. In contrast to the test of perception of facial emotion anterior and posterior patients performed at a similar level on both the immediate and delayed matching conditions (Table 7). This would suggest that the anterior and posterior groups do not differ in their ability to perceive and remember particular faces.

Table 8 does, however, show significant correlations between facial identity and facial emotional recognition scores. Therefore, to further evaluate the possibility that facial emotional and identity recognition are dissociable functions, the anterior and posterior groups' abilities to perceive facially expressed emotion were compared in a one-way analysis of covariance using total facial identity recognition scores as the covariate. The mean facial emotion score after adjusting for the covariate (facial identity score) was 16.8 faces correct in the anterior group, and 22.9 faces correct in the posterior group. However, this superiority in adjusted mean score of the posterior group fell short of statistical significance [ $F(1,15)=3.84$ ;  $p=0.07$ ].

The facial identity recognition test was incorporated at a later stage of data collection and there were only 8 anterior and 10 posterior patients suitable for this analysis. Furthermore, ceiling effects in the distribution of facial identity recognition scores raise significant doubt about the validity of this covariance analysis. However, another test employing facial stimuli was also administered. Like the facial identity test, the facial recognition memory test was not significantly impaired in the anterior group (Table 7), and it correlated with facial emotional recognition scores (Table 8). Since 13 anterior and 16 posterior patients completed the facial recognition memory test, a second analysis of covariance was performed using facial recognition memory test scores as the

covariate, and the anterior group was still significantly impaired with respect to patients with posterior lesions [ $F(1,24)=5.87$ ;  $p=0.023$ ].

	<u>Anterior</u> <u>Group</u>	<u>Posterior</u> <u>Group</u>	<u>t-value</u>	<u>p value</u>
<b>Face Recognition</b>				
0 secs delay	8.8	8.4	0.86	0.39
4 secs delay	7.3	8.3	1.47	0.16
<b>Memory</b>				
Face recognition	14.1	15.8	0.86	0.50
Word recognition	13.5	17.8	1.72	0.10
Benton Visual Retention	5.0	5.3	0.36	0.75
<b>Cognitive tests</b>				
Verbal IQ	81.2	92.8	2.09	0.046
Performance IQ	85.1	89.9	0.97	0.34
Perseverative errors (WCST)	71.0	41.0	2.93	0.007

Table 7

Comparison of anterior and posterior groups on tests of cognition, memory and facial identity perception

Table 7 also shows that the proportion of perseverative errors on the Wisconsin Card Sorting Test was significantly lower in the anterior group, as was mean Verbal IQ. Moreover, within the group of patients who had either an anterior or a posterior lesion, facial emotion perception scores correlated significantly with word recognition memory, and near significantly with Performance IQ (Table 8). Therefore in a further analysis Performance IQ, Verbal IQ, perseverative errors, word and face recognition memory scores were partialled out. Though a significant association between these 5 covariates and facial emotion test scores was found [ $F(5,16)=10.13$ ;  $p=0.001$ ], the anterior group

(N=10) were still impaired relative to the posterior group (N=13) for perception of facial emotion remains [ $F(1,16)=6.6$ ;  $p=0.02$ ].

	<u>Total</u> <u>Score</u>	<u>Face-</u> <u>Face</u>	<u>Face-</u> <u>Word</u>	<u>Delay</u> <u>0 Sec</u>	<u>Delay</u> <u>4 Sec</u>
Face Identity Recognition	0.53*	0.55*	0.43	0.37	0.59**
Face Recognition Memory	0.57**	0.51**	0.51**	0.52*	0.53**
Performance IQ	0.30	0.26	0.27	0.20	0.30
Perseverative Errors (WCST)	-0.50**	-0.48*	-0.37	-0.43*	-0.47*
Verbal IQ	0.06	0.02	0.08	0.09	0.03
Word Recognition Memory	0.55**	0.40*	0.59**	0.58**	0.45*

Table 8

Correlations between measures of perception of facially expressed emotion and indices of perceptual/cognitive deficit (\*\*- $p<0.01$ ; \*- $p<0.05$ )

#### Perception of type of facial emotion and lesion site

There is another potential way of establishing a special role for frontal regions in the regulation of emotional behaviour. The case for a specific emotional disturbance after anterior damage would be strengthened if we could show that anterior damage selectively impaired the patient's ability to perceive some emotions, leaving other emotional perceptions intact (or at least, relatively spared). In particular it has often been observed that after anterior

damage patients tend to be mildly euphoric (Hecaen, 1964). We might therefore predict that ability to perceive happy faces would be relatively preserved, or even enhanced in frontal patients. Conversely, ability to perceive negative emotional states would be selectively impaired. In the statistical model, we would find an interaction between emotional type and lesion site. To assess this possibility a 3x6 analysis of variance was performed with lesion site (anterior v posterior v control) as one factor, and repeated measures for emotional type (happy v sad v angry v fear v disgust v surprise).

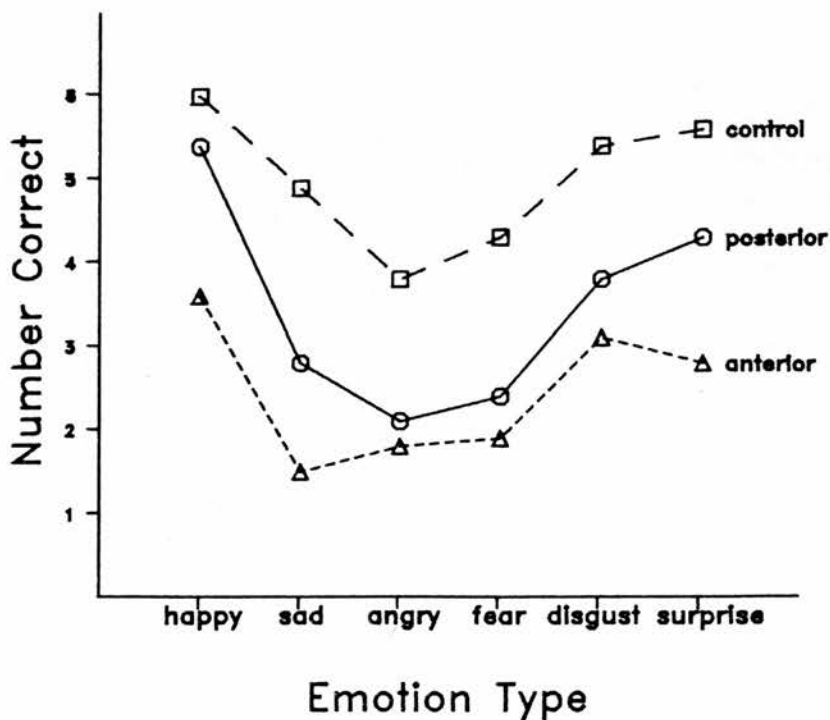


Figure 5

Mean scores for perception of different types of facially expressed emotion in control, anterior and posterior groups

Using the SPSSX multivariate repeated measures analysis of variance procedure, Wilks Lambda for the type of emotion factor was 0.204. This multivariate equivalent of the F test gives an approximate F-value of 25.73;  $df=5,33$ ; and  $p<0.001$ . The effect of emotion type is displayed graphically in figure 5, which shows that all three groups of subjects had most difficulty with the sad, angry and fearful faces. It would seem that the anterior lesion group was generally impaired relative to the posteriors and the controls for all types of emotion, as there was no interaction between lesion site and type of emotion [Wilks Lambda=0.687; Approximate  $F(10,66)=1.36$ ;  $p=0.22$ ]. While a selective impairment in the perception of certain types of emotion would add weight to the hypothesis that a specifically emotional disorder accounts for the greater anterior impairment on the perception of facial emotion test, the lack of evidence of selective deficit does not contradict the hypothesis. It is quite conceivable that all emotional behaviours tend to be affected by large frontal lesions.

### Conclusions

1. While both anterior and posterior cerebral damage impaired performance on the test of perception of emotion expressed in the face, the deficit was most marked in patients with frontal lesions.
2. Anterior and posterior groups differ with regard to size and type of lesion, but after partially controlling for these variables the anterior group still showed significantly more deficit than the posterior group. However when anterior and posterior groups were matched for degree of bilateral damage, only a non-significant tendency to greater anterior impairment remained.



3. To establish the impairment of the anterior lesion group as a selective loss of visual-emotional perception it is necessary to exclude the possibility that deficit in facial emotion perception is due to a general perceptual/cognitive deficit. While the results are most consistent with the possibility of selective impairment in emotional perceptions after anterior damage, the evidence is insufficient for a firm conclusion on this point.
4. There were insufficient cases to allow a meaningful comparison between different divisions within the anterior, or posterior, sites. There was weak evidence suggesting that the basal ganglia have a special role in the perception of facially expressed emotion. An expected relationship between medial temporal damage and deficits in perception of emotions was not found.
5. There was no evidence suggesting that ability to perceive positive emotion was selectively preserved after anterior damage.

#### 4.2. Test of Perception of Emotion Expressed in Speech and Text

As explained above, each patient was presented with tape recorded utterances, and written sentences. Using three groups of normal raters these verbal stimuli had been classified as expressing emotion in tone of voice, or in both tone of voice and semantic content, or in semantic content alone. The patient identified the emotion by matching (a) to the verbal labels; happy, sad, angry, neutral, and surprised or (b) to one of 5 pictures of standard emotional faces. The relevant measures derived from this test are:

- Total number of emotional stimuli correct
- Number of spoken sentences correctly identified where emotion was expressed solely in **tone** of voice.
- Total correct for spoken sentences where emotion was expressed in **tone and content**.
- Number correct for written test sentences where emotion was expressed in the verbal-semantic **content**.
- Number of (a) happy, (b) sad, (c) angry, and (d) neutral sentences correctly identified.
- Number of sentences correctly identified when (a) matching to the verbal labels and (b) matching to 5 standard emotional faces.

In a 3x2 analysis of variance total scores on the test of perception of emotion conveyed in speech and text was the dependent variable; with lesion site (control v anterior v posterior), and sex as factors. Only lesion site was significant [ $F(2,36)=27.86$ ;  $p<0.0001$ ]. As neither sex [ $F(1,36)=0.02$ ;  $p=0.89$ ], nor the sex by site interaction [ $F(2,36)=0.10$ ;  $p=0.91$ ] achieved significance, the 23 males and the 19 females are treated as identical for subsequent analyses.

<u>Score Range</u>	<u>Anterior Group</u>	<u>Posterior Group</u>	<u>Control Group</u>
65-60		1	5
59-54		3	1
53-48	1	2	4
47-42	2	8	
41-36	4	3	
35-30	1		
29-24	3		
23-18	2		
17-12	1		
11-6			
5-0	1		

Table 9

Total scores on the test of verbally expressed emotion for the control, anterior and posterior groups.

Table 9 gives total scores for patients in the anterior ( $N=15$ ), posterior ( $N=17$ ), and control ( $N=10$ ) groups, and in a one-way analysis of variance a highly significant group effect was found [ $F(2,39)=30.01$ ;  $p<0.0001$ ]. Post hoc testing using the Scheffe procedure shows the control group were significantly superior to both the posterior, and the anterior lesion groups. Therefore, as with the test of visual perception of emotion in faces, any brain damage impairs performance on the test of perception of verbally expressed emotion.

Furthermore, the anterior lesion group showed significantly greater impairment than the posterior lesion group. This greater anterior deficit appears to support the hypothesis that the frontal lobes are specialised for the perception of emotion, but we must, once again, consider alternative ways of explaining the relatively greater difficulty shown by the anterior lesion group.

### **Perception of emotion expressed in speech and text, with lesion size and location**

**Lesion size and aetiology:** We have seen that lesion size was larger in the anterior lesion group. Therefore anterior and posterior groups were compared for perception of emotion conveyed in speech and text, after excluding patients with frontal lobectomy, and adjustments were made for pre-operative lesion volume in a one-way analysis of covariance. There were 9 anterior and 17 posterior patients available for this analysis. Lesion size did significantly influence test scores [ $F(1,22)=8.82$ ;  $p=0.007$ ], but after this effect was partialled out the greater anterior lesion deficit remained [ $F(1,22)=21.43$ ;  $p<0.001$ ].

The anterior and posterior groups were then compared in the subgroups matched for lesion aetiology. The influence of lesion size [ $F(1,13)=2.95$ ;  $p=0.11$ ] was also partialled out, but again the 9 anteriors showed significantly greater impairment than the 7 posteriors [ $F(1,13)=9.05$ ;  $p=0.01$ ]. Finally, the 8 anterior patients with bilateral lesions performed significantly less well than the 7 anterior unilateral lesion subgroup (Mann-Whitney  $U=8.5$ ;  $p=0.021$ ). Nonetheless, after anterior and posterior groups were matched for degree of bilateral damage, the anterior lesion group ( $N=8$ ) remained relatively more impaired than the 17 posteriors [ $F(1,23)=13.81$ ;  $p<0.001$ ].

**Lesion location:** We have previously commented that in this present sample it was not possible to meaningfully compare dorsolateral and orbital frontal lesions, or temporal and parietal lesions. Nevertheless we were able to make a limited assessment of the contribution of medial temporal subcortical structures and the basal ganglia to performance on the different emotional tests. The 18 patients with damage to medial temporal structures were compared with the 28 remaining cases, and again, there was no difference between groups on total scores for perception of verbally expressed emotion ( $t=0.03$ ;  $df=44$ ;  $p=0.98$ ). On the other hand, 23 patients with lesions encroaching on the basal ganglia were compared with the 23 other cases. Though this analysis indicated that extension of the lesion into basal ganglia structures was associated with a greater impairment of emotional perception ( $t=2.45$ ;  $df=44$ ;  $p=0.018$ ), the potential confounding influences of lesion size and lesion depth were not controlled. Next, patients who had not had a lobectomy and who had damage to only one subcortical structure were considered. No significant difference in perception of verbally expressed emotion was demonstrated when the 3 patients with basal ganglia lesions were contrasted with the 6 patients who had either medial temporal or thalamic damage (Mann-Whitney  $U=5$ ;  $p=0.57$ ).

#### Perception of emotion in speech and text, lesion site and cognitive function

Again in agreement with the results from the test of perception of facial emotion, the anterior lesion group was impaired relative to the posterior group on all measures derived from the test of verbally expressed emotion (Table 10). This pattern was seen whether emotion was expressed in tone of voice, or in tone plus semantic content, or in content alone. Furthermore, both matching to faces and matching to words significantly more impaired in the anterior group.

Therefore, neither manipulation of test stimulus characteristics, nor change of response requirements, yielded evidence that the anterior deficit was due to cognitive-perceptual factors.

	<u>Anterior Group</u>	<u>Posterior Group</u>	<u>t-value</u>	<u>p value</u>
<b>Stimulus Manipulation</b>				
Tone	8.5	13.2	3.13	0.004
Content	12.3	19.0	4.18	0.001
Tone and Content	9.9	15.2	4.16	0.001
<b>Response Manipulation</b>				
Face match	14.6	22.3	3.85	0.001
Word match	16.1	25.1	4.00	0.001

Table 10

Mean scores for the anterior and posterior groups on some measures derived from the test of perception of emotion expressed in speech and text.

Test scores for perception of facially expressed emotion were adjusted for facial identity recognition, as well as memory for faces. It would have been desirable to have administered comparable tests of perception of non-emotional auditory, and verbal, stimuli constructed to correspond to the test of perception of emotion expressed in speech and text. The Seashore Test of Musical Ability (Milner, 1962), or tests of perception of non-affective speech intonation (Weintraub *et al*, 1981) might have been incorporated. However such tests were not given because it was feared that this might exceed the amount of assessment the patient could tolerate. Nevertheless, other cognitive tests were administered, and Table 7 shows that the anterior lesion group was

impaired relative to the posteriors for Verbal IQ, and perseverative errors on the Wisconsin Card Sorting Test. Furthermore, within the group of patients who had either an anterior or a posterior lesion, verbal emotion perception scores correlated significantly with several measures of perceptual/cognitive deficit (Table 11). Therefore, scores of the anterior and posterior lesion groups for the test of perception of verbal emotion were compared in a one-way analysis of covariance, with adjustments made for Performance IQ, Verbal IQ, perseverative errors, word and face recognition memory. The 12 anterior patients' scores were still significantly more impaired than the 13 posterior patients' scores, after this adjustment [ $F(1,18)=4.49$ ;  $p=0.04$ ].

	<u>Total Score</u>	<u>Verbal- Face</u>	<u>Verbal- Word</u>	<u>Content</u>	<u>Tone</u>
Verbal IQ	0.31	0.16	0.37*	0.32	0.21
Word Recognition Memory	0.55**	0.47**	0.51**	0.52**	0.41*
Performance IQ	0.39*	0.44*	0.25	0.32	0.37*
Face Recognition Memory	0.51**	0.57***	0.35	0.44*	0.35
Perseverative Errors (WCST)	-0.49**	-0.45*	-0.42*	-0.44*	-0.36

Table 11

Correlations between measures of perception of verbally expressed emotion and indices of perceptual/cognitive deficit (\*\*\*- $p<0.001$ ; \*\*- $p<0.01$ ; \*- $p<0.05$ )

### Perception of type of emotion expressed in speech and text, and lesion site

In the test of verbally expressed emotion 20 sentences were happy, 18 were sad, 23 were angry and 11 were neutral. To statistically assess the possibility that certain emotions are more easily perceived by all groups it is necessary that the maximum score for each emotional type is uniform. Therefore for a 3x3 repeated measures multivariate analysis of variance the happy, sad, and angry raw scores of each patient were converted into percent correct happy, sad and angry scores. The type of emotion factor was significant [Wilks Lambda=0.829; Approximate  $F(2,38)=3.93$ ;  $p=0.03$ ]. Using post hoc univariate contrasts it was found that more errors were made with the angry sentences than with either happy or sad sentences. Figure 6 raises the possibility that, compared with controls and the posterior lesion patients, the anterior lesion group had particular difficulty in perceiving sad and angry emotion. However the site by emotion type interaction fell far short of significance [Wilks Lambda=0.863; Approximate  $F(4,78)=1.45$ ;  $p=0.25$ ]. Therefore, as with the test of perception of facially expressed emotion, there was no support for the hypothesis that the ability to perceive positive emotion was selectively spared after anterior cerebral damage.

### Conclusions

1. Perception of emotion in speech and text was impaired after anterior and posterior lesions. Frontal lesions resulted in most severe deficit.
2. The greater deficit in the anterior lesion group remained after controlling lesion size, lesion aetiology, and degree of bilateral damage.
3. The frontal deficit did not appear to be wholly due to perceptual/cognitive impairment, but perceptual/cognitive factors were only partially controlled.

4. There were indications that the basal ganglia have a special role in the perception of emotion conveyed in speech and text. Again, the expected relationship between medial temporal damage and impaired emotional perception was not found.
5. There was no evidence that ability to perceive positive emotion was selectively impaired after anterior damage.

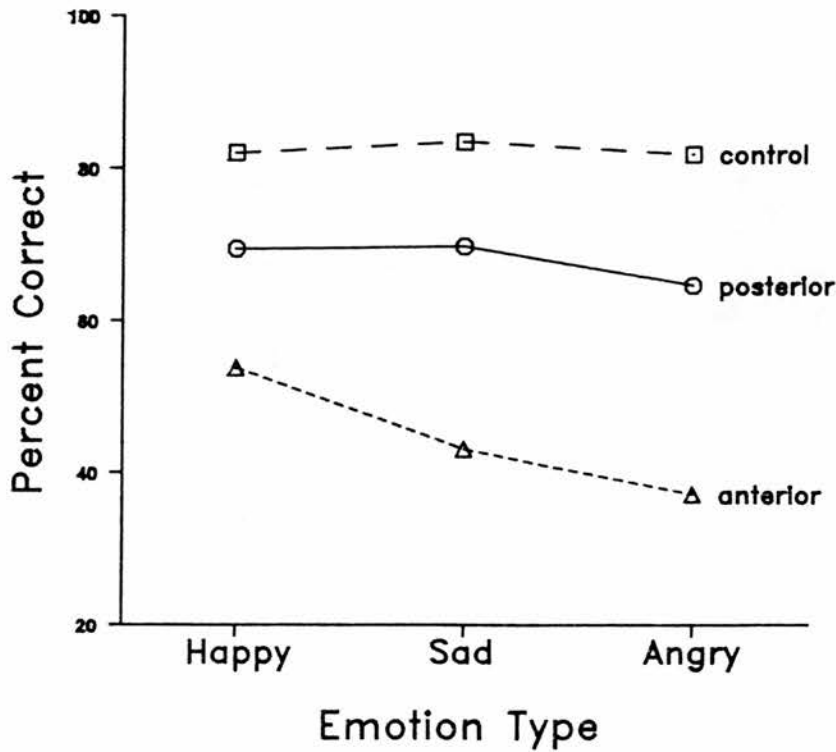


Figure 6

Mean scores on the test of perception of verbally expressed emotion for different types of emotion in control, anterior, and posterior groups



### 4.3. Discussion

On both tests of perception of emotion, patients with anterior lesions were significantly inferior to the posterior group. This greater impairment following damage to anterior regions did not appear to be easily attributable to the tendency for anterior lesions to be larger than, or of a different aetiology to, the posterior lesions. However, when anterior and posterior groups were matched for degree of bilateral damage for the test of perception of facially (but not verbally) expressed emotion, the tendency for greater anterior deficit fell short of significance. Furthermore, Kolb and Taylor (1981) did not find significantly greater impairment in their anterior lesion groups on a similar test, but in that study mean scores were notably lower in the right and left frontal groups.

The anterior group was impaired relative to the controls and posterior groups for perception of all types of emotion in both tests of perception of emotion. This general impairment in the perception of all types of emotion is clearly consistent with the view that the frontal lobes are specialised for the regulation of all emotional processes. Evidence for a specifically emotional deficit, revealing more of the mechanism of emotion, would be gained if certain emotions were selectively more impaired after frontal damage. In particular, given the frequent reports of euphoric symptoms after frontal damage, it might have been expected that the perception of negative affective states would be especially disordered following frontal damage. However there was no significant interaction between emotional type and lesion site for either of the tests of perception of emotion.

Comparative functional neuroanatomy of visual perception was one of the main guides for the experimental design employed in the present study.

Neuroanatomical, electrophysiological and lesion studies all suggest that in the primate brain the physical features of emotionally significant stimuli are first analysed in posterior neural regions before the affective relevance or meaning of the stimulus is evaluated in more anterior limbic cortical-subcortical systems. The present data is consistent with this model in three respects. First, this model predicts that both posterior and anterior damage will affect emotional perceptions. Posterior lesions will impair emotional perceptions because of a general perceptual disorder, and anterior lesions impair emotional perceptions because of a specifically emotional dysfunction. In confirmation of this, both posterior and anterior lesion groups were impaired relative to the controls on both of the tests of emotional perception. Secondly, we would expect to find an association between severity of general sensori-motor-cognitive deficit and performance on the perception of emotion tests. This again was found. For example, a significant association was found between facial emotional and facial identity perception. Thirdly, when level of general sensori-motor-cognitive deficit was statistically controlled there were strong indications that the greater anterior deficit remained on both of the tests of emotional perception. Nevertheless in one analysis perception of facial identity and facial emotion scores were considered together, but only a non-significant trend for greater anterior deficit was demonstrated. However in other studies of normals (Ley and Bryden, 1979) and of brain damaged patients (Etcoff, 1984; Dekosky *et al.*, 1980) there have been indications of a dissociation between facial identity and emotional perception.

One important prediction of the model derived from comparative research was not confirmed. The amygdala and the hippocampus are thought to create an interface between general sensori-motor processes and the limbic system. On this view, the amygdala is centrally involved in the process where sensory

stimuli are invested with emotional significance. It had therefore been expected that damage to medial temporal subcortical structures would impair performance on both test of emotional perception. Though this effect was not found, there is ample evidence from other studies of emotional changes following amygdalar lesions. Presumably, other test procedures may be better suited to reveal these emotional disorders.

## CHAPTER 5

### THE ROLE OF THE FRONTAL LOBES IN THE EXPRESSION OF EMOTION

Spontaneous emotional responses were investigated in a failure test situation, and the patients' capacity to voluntarily produce facial emotional expressions was also studied. If the frontal lobes are specialised for the production of emotional responses the anterior group would be expected to show selective deficit on one or both of these experimental tasks.

#### 5.1. Spontaneous Reactions in a Failure Situation

The patients' face and head movements were video recorded while a modified version of the Wisconsin Card Sorting Test was administered. Briefly, the patient was required to sort response cards under key cards according to certain rules. He was not explicitly told what the sorting rule was. After sorting each card he was told whether he made a correct, or an incorrect, sort. The main relevant measures taken were:

- FACS (Ekman and Friesen, 1978) was used to quantify the patient's facial movements immediately following the first 10 occasions when he was told he was wrong. In addition to measuring facial movement, total head movements, comments, and vocalisations were noted
- Qualitative ratings of positive and negative emotional reactions to failure were made for each of the 10 reactions to being told he was incorrect. Qualitative ratings of positive and negative emotional reactions were also made for each of the 10 first correct card sortings.
- 40 cards were sorted, and for all patients, 20 sorts were said to be wrong. Some attempt having been made to equate the difficulty of the failure situation, patients had to indicate their inner subjective feeling response to the failure task.
- Heart-rate was used to provide an autonomic index of emotional reaction. Baseline heart-rate, heart-rate when correct, and heart-rate when wrong were measured.

**Selectively reduced spontaneous facial movement in the anterior lesion group**

In a 3x2 analysis of variance total number of FACS action units produced in the failure situation was the dependent variable; with lesion group, and sex as factors. The number of action units produced was associated with lesion group [ $F(2,32)=14.08$ ;  $p<0.0001$ ]. Neither sex [ $F(1,32)=0.06$ ], nor the sex by site interaction [ $F(2,32)=0.71$ ] attained significance. Therefore in subsequent analyses the 21 male and 17 female subjects are grouped together.

<u>Action Units</u>	<u>Anterior Group</u>	<u>Posterior Group</u>	<u>Control Group</u>
36-33			1
32-29		1	
28-25		3	3
24-21	1	3	1
20-17	1	5	1
16-13	1	3	2
12-9	6		
8-5	3	1	
4-0	2		

Table 12

Total number of FACS action units produced during 10 reactions to failure in the anterior, posterior and control groups

Table 12 suggests that there is a selective reduction of facial movement in the anterior lesion group. This was confirmed by a one-way analysis of variance, where total FACS action units elicited by the failure situation was the dependent variable. Post hoc testing using the Scheffe procedure showed that patients with anterior lesions ( $N=14$ ) produced significantly fewer facial action

units than the control (N=8) or the posterior (N=16) lesion groups, but there was no difference between the controls and the patients with posterior lesions.

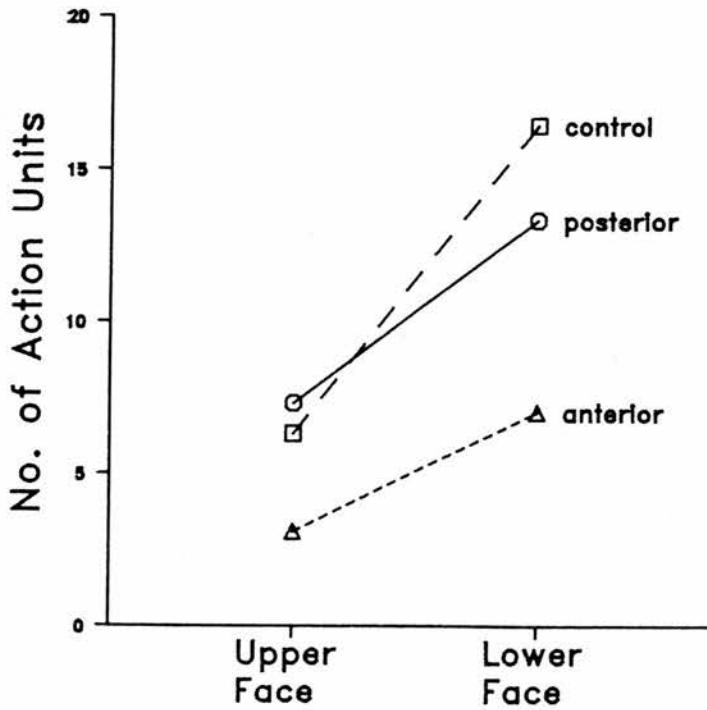


Figure 7

Upper and lower face spontaneous movements in the failure situation; anterior, posterior, and control groups compared.

The upper part of the face and forehead are under direct bilateral cortical control. Though there is an indirect corticobulbar pathway to reticular interneurons which projects to the facial nerve nucleus, direct cortical control of the lower face is unilateral and crossed (Rinn, 1984). It is therefore desirable to consider upper and lower face spontaneous movement separately. A

repeated measures analysis of variance was performed with number of FACS action units as the dependent variable. Lesion site (anterior v posterior v control) was the between subjects factor; face (upper v lower) was the within subjects factor. As in previous analyses, the effect of lesion site was significant [ $F(2,35)=14.68$ ;  $p<0.0001$ ]. Also, in all groups, significantly more lower face movements were elicited in the failure situation [ $F(1,35)=33.47$ ;  $p<0.0001$ ]. Of more relevance to the present study the face by site interaction fell short of significance [ $F(2,35)=2.17$ ;  $p=0.13$ ]. However this non-significant trend did not indicate that upper and lower face movements were differentially affected by anterior and posterior damage. Figure 7 would suggest that the non-significant interaction is due to the higher proportion of lower face movements in controls, relative to both lesion groups.

	<u>Anterior</u> <u>Group</u>	<u>Posterior</u> <u>Group</u>	<u>Control</u> <u>Group</u>
Upper Face	3.1	7.3	6.3
Lower Face	7.0	13.3	16.4
Head Movements	3.4	6.6	4.8
Comments	0.9	3.7	2.9
Vocalisations	1.6	1.7	1.5

Table 13

Mean number of component movements produced by anterior, posterior, and control groups after being told an incorrect card sort had been made.

Table 13 suggests that reduced response may not have been confined to facial movements. Frontal patients made significantly fewer head movements

than posteriors ( $t=2.77$ ;  $p=0.01$ ), but not controls ( $t=0.99$ ;  $p=0.33$ ). Comments were less common in the anterior group relative to posterior patients ( $U=64.5$ ;  $p=0.47$ ), but not controls ( $U=38.5$ ;  $p=0.24$ ). Frequency of spontaneous vocalisations was similar across the three groups.

### **Spontaneous facial movement with lesion size and location**

**Lesion size and aetiology:** It may be that the larger lesion size in the anterior group accounts for their selectively impoverished facial expression. In a one-way analysis of covariance, patients with frontal lobectomy were excluded, and the influence of total lesion volume was partialled out. After adjustments were made for the non-significant effect of lesion volume [ $F(1,21)=1.63$ ;  $p=0.22$ ], the selective reduction of facial expression in the 9 patients with anterior lesions remained [ $F(1,21)=13.94$ ;  $p<0.001$ ]. The groups were next matched for lesion type by excluding patients who had a meningioma, or a metastasis or an AVM, or stroke, or frontal lobectomy; and again total lesion volume was included as a covariate. The effect of lesion size was not significant [ $F(1,11)=1.45$ ;  $p=0.25$ ], and as before the anterior group ( $N=9$ ) was still found to produce significantly fewer facial actions than the 5 posterior patients [ $F(1,11)=12.15$ ;  $p=0.005$ ]. It would seem that lesion site is more important than lesion size in determining the poverty of facial expression in this sample. This conclusion is given further weight when we note that patients with (sometimes large) posterior lesions made as many facial responses as the controls i.e. a large lesion size does not inevitably induce impoverished facial expression.

Poverty of facial expression was not related to degree of bilateral damage in any obvious way. Patients with ( $N=8$ ) and without ( $N=6$ ) bilateral anterior damage produced the same number of spontaneous action units (Mann-Whitney  $U=22.5$ ;  $p=0.85$ ). Furthermore, the anterior and posterior groups



were also matched for degree of bilateral involvement. There were 15 posterior patients with a unilateral lesion plus one patient with bilateral damage. In the anterior group 6 had a unilateral lesion and one had bilateral involvement. However this matching did not eliminate the selective reduction in facial expression in the anterior group ( $t=4.52$ ;  $p<0.0001$ ).

**Lesion location:** Patients with ( $N=13$ ), and without ( $N=28$ ), medial temporal damage were compared but no significant difference was found for total number of facial action units produced ( $t=0.59$ ;  $p=0.56$ ). Patients with ( $N=18$ ) and without ( $N=23$ ) damage to the basal ganglia did differ significantly ( $t=3.74$ ;  $p<0.001$ ). However in this comparison extent of subcortical damage is uncontrolled, as 15 patients in the group without damage to the basal ganglia had comparatively superficial lesions with no associated subcortical damage. But deeper lesions may cause more severe deficit (Lishman, 1968), so it might be argued that reduction of facial expression is related to degree of subcortical damage *per se*, rather than to disturbance of special functions performed by basal ganglia structures. Therefore patients whose lesion invaded only one group of subcortical structures were considered. The 3 patients with damage solely to the basal ganglia (caudate nucleus, and/or putamen, and/or globus pallidus, but without frontal lobectomy) were contrasted with the 5 patients who had only medial temporal or only thalamic damage. In this comparison patients with damage to the basal ganglia tended to produce significantly fewer facial actions than patients with subcortical damage sparing the basal ganglia (Mann-Whitney  $U=0.5$ ;  $p=0.057$ ).

#### The reduced facial response to failure as a primary motor deficit

A link between damage to the striatum and reduced spontaneous facial movement has long been noted in patients with Parkinsons' disease (Walton,

1981). It has been thought that this poverty of facial expression is but one manifestation of a general impairment of motor functions. Furthermore damage to the frontal lobes, including premotor cortex, can also result in motor disorders such as hypokinesia and limb-kinetic apraxia (Freund and Hummelsheim, 1985; Luria, 1966). Furthermore, the frontal cortical fields project to the striatum (Brodal, 1981; Kemp and Powell, 1970). Perhaps damage to fronto-striatal systems produces a predominantly facial-motor disorder. Certainly, in some respects the data were consistent with this possibility (see tables 14 & 15).

	Reaction Absent	Reaction Present
Anterior Group	60	80
Posterior Group	33	127
Control Group	13	67

Table 14

Relative frequency of occasions when a measureable facial reaction was noted. Anteriors produced significantly fewer reactions than posteriors or controls.

The selective reduction in the number of action units elicited was due to two effects. First, the anterior patients reacted less frequently. In table 14 the 10 reactions of the 14 anterior patients who completed the card sorting task are grouped together. In only 57% of these 140 reactions was a quantifiable

facial movement noted. Quantifiable facial movement was significantly more frequent in the posterior lesion group, occurring on 79% of the 160 occasions ( $\chi^2=17.3$ ;  $df=1$ ;  $p<0.001$ ). Compared with the anterior lesion patients, controls also made a significantly higher proportion (84%) of measureable facial responses ( $\chi^2=16.2$ ;  $df=1$ ;  $p<0.001$ ). Secondly, when anterior patients did make a facial response, their reactions tended to be impoverished, involving fewer facial-motor components. In table 15 we consider only those occasions when patients produced a measurable facial response. While 44 (55%) of the reactions of patients with anterior lesions involved only one FACS action unit, only 30 (24%) of the reactions of the posterior group consisted of one action unit. In contrast, relatively more reactions in the posterior group involved 4 or more action units. These distributions were significantly different ( $\chi^2=23.1$ ;  $df=1$ ;  $p<0.001$ ). Similarly, 25% of the reactions of controls involved a single action unit, and the distribution of action units per reaction was also significantly different for controls and patients with anterior lesions ( $\chi^2=18.5$ ;  $df=3$ ;  $p<0.001$ ).

This reduction of facial movement might be due to a general disorder of facial movement. Perhaps all facial movement is impoverished or reduced, and we have suggested that this general movement disorder may be the result of damage to fronto-striatal neural systems. In point of fact, striatal lesions produce complex deficits (Divac and Oberg, 1979); which implies that either (a) other functional deficits co-exist with motor disorders and/or (b) that striatal motor disorders are usually more complex and of a "higher order" than was first thought. This suggests a second possible explanation for the impoverished facial expression in patients with anterior lesions. The deficit might not be a "simple" motor disorder, it may be a more complex emotional-motor disturbance, with other non-emotional movements relatively preserved. Certainly, we know that emotional reactions have a "pre-wired" or innate motor

component. Such innate motor synergisms might be characterised as being simultaneously motoric and emotional. Furthermore, lesions in the globus pallidus severely disrupt the species-specific "trump" display of the squirrel monkey (MacLean, 1978). Also, we have argued that affective and general sensori-motor functions are integrated in the frontal lobes, which project anatomically to the neostriatum. Convergence of motor commands and affective information in certain cortical fields (Nauta, 1971) and in striatal sites (Heimer *et al.*, 1982) would lead to integrated affective-motor functions. This would in turn suggest that damage to the regions integrating motor commands and affective information produces a class of movement disorders which are simultaneously and irreducibly motor-emotional.

	1 AU	2 AU	3 AU	4+ AU
Anterior Group	44	19	10	7
Posterior Group	30	41	24	32
Control Group	17	14	20	16

Table 15

Number of times patients produced 1,2,3 or 4 FACS action units (AU) in response to being wrong. Over half of the reactions in the anterior group involved only one AU, while only a quarter of the reactions of the other groups consisted of one AU.

In order to distinguish these two possible explanations for impoverished spontaneous facial expression, tests of the patient's capacity to make non-emotional facial movements were included in the test battery. Upper face movements accompanying 30 seconds of spontaneous speech were counted. The patient was also required to reproduce single facial movements in sequences of 3. On both measures the anterior lesion group were significantly impaired relative to the posteriors (Table 16). Therefore the poverty of facial emotional expression in the anterior group occurs within the context of a general deficit in non-emotional facial movement. However we cannot yet conclude that the poverty of spontaneous facial reaction is solely due to a general movement disorder. It may be that patients with anterior damage suffer from both a general facial movement disorder and a deficit in the production of specifically emotional-facial movements.

	<u>Anterior</u> <u>Group</u>	<u>Posterior</u> <u>Group</u>	<u>t-value</u>	<u>p</u>
Upper face movements in speech	3.1	5.3	2.06	0.05
Imitation of face movement	13.8	6.0	2.41	0.03

Table 16

Reduced spontaneous facial expression occurs in the context of generally reduced facial movements.

To examine this alternative total FACS action units produced by anterior and posterior patients in the failure situation were compared in a one-way analysis of covariance. The covariates were upper face motion during speech,

and total errors made imitating facial movement sequences (Table 16). After adjustments were made for these two non-emotional movement covariates the anterior group (N=9) still made significantly fewer facial motions than posteriors (N=13) in the failure situation [ $F(1,18)=11.98$ ;  $p=0.003$ ]. This may indicate that in the failure situation reduced facial expression of the anterior group co-exists with, but is not wholly reducible to, a general facial movement disorder. Indeed, the two covariates considered together were not significantly related to amount of spontaneous response to failure [ $F(2,28)=2.17$ ;  $p=0.15$ ], which would provide some further support for the hypothesis that emotional and non-emotional facial movements are dissociable functions.

#### The reduced facial response to failure as a primary cognitive/attentional disorder

As the lack of facial response in the anterior group is not wholly due to a general motor disorder we must next ask whether cognitive factors are responsible. Table 7 shows that the anterior group made significantly more perseverative errors on card sorting. The tendency for frontal patients to continue sorting cards according to a rule that is wrong, may indicate that the frontal patients were not as aware of having failed as were the posteriors or controls. Perhaps their reduced facial response to failure merely reflects a lack of awareness of errors and loss of insight.

To test this hypothesis anterior and posterior lesion groups were compared in an analysis of covariance, where total spontaneous FACS action units was the dependent variable and proportion of perseverative errors was the covariate. If lack of awareness of error accounts for both the perseverative tendency and the reduced facial reaction to failure it is predicted that the group effect would no longer achieve significance. However 14 the anterior

patients were still significantly impaired in emotional expression relative to the 15 posteriors [ $F(1,26)=16.27$ ;  $p<0.001$ ], even though there was a near significant association between the covariate and spontaneous facial reaction [ $F(1,26)=3.86$ ;  $p=0.06$ ].

Table 7 shows that Verbal IQ is significantly lower in the anterior lesion group and there is also a trend for greater anterior deficit in word recognition memory. It is therefore possible that a better index of loss of insight in the anterior group would be gained through combining the effects of perseverative errors, Verbal IQ, and word recognition memory scores as covariates in an analysis of covariance. After adjustments were made for the joint influence of these covariates [ $F(3,20)=0.66$ ;  $p=0.59$ ], the 12 anterior patients again showed significantly reduced facial response relative to the 13 posteriors [ $F(1,20)=15.27$ ;  $p<0.001$ ].

It is concluded that while cognitive factors may partially account for the lack of facial expression in the anterior patients, but a cognitive explanation is not sufficient on its own to account for the effects of anterior lesions on emotional expression.

#### Reduced facial response as a primary emotional deficit

It has been suggested that the reduced expressiveness of the anterior group is **not** simply attributable to lesion size, or lesion type, or a primary generalised motor deficit, or a primary cognitive/attentional disorder. However this conclusion is largely based on negative proof, which suffers from a fundamental limitation. For example, we concluded that the frontal lobes are specialised for facial-emotional expression, because an anterior deficit for spontaneous facial expression was still evident after adjusting facial reaction

scores with two measures of non-emotional facial movement. The problem is that control for non-emotional facial movement disorder can never be complete or exhaustive. There may be several types of non-emotional facial movement disorder associated with frontal damage. Indeed, the two present measures of non-emotional facial movement (viz. upper face speech markers and imitation of meaningless facial movements) were not significantly related ( $r=-0.15$ ;  $df=36$ ;  $p=0.35$ ). It is therefore always possible that adjustments to facial reaction scores with other measures of non-emotional movement would eliminate the selective anterior deficit in spontaneous emotional expression.

Since negative proof cannot, on its own, provide compelling evidence for an emotional specialisation of the frontal lobes, positive evidence should be sought where possible. To gain positive proof, in this investigation multiple measures of emotional response were made in the failure situation. Qualitative ratings were made of the video recordings of patients' reactions to being told they had sorted a card correctly or incorrectly. The patient also rated their own subjective reaction to the card sorting situation. To obtain an autonomic index of the patients' emotional response, various heart-rate measures were made. If an anterior deficit was noted on all three additional emotional measures we would have strong positive evidence for an emotional specialisation in the frontal lobes.

**Qualitative ratings:** Since patients with anterior lesions had lower ratings than the posterior patients on all 4 of the qualitative indices of reaction (Table 17), a general suppression of all emotional responses in the anterior group is suggested, but the anteriors' scores were only significantly lower than posteriors and controls on positive reaction after wrong sorts. It is also noteworthy that table 17 provides no evidence that positive emotional states were spared more than negative emotional states after frontal damage.



Though these qualitative ratings of the patient's reactions in the card sorting task give some independent support for the conclusions gained with quantitative ratings (using FACS), the quantitative and qualitative indices of facial reaction are not fully independent measures. Both qualitative and quantitative measures of emotional reaction are derived from the same video-recordings, and so might inevitably give similar results. However the patients also described their subjective reaction to the task. This self-report measure of emotional response was derived separately from the ratings of facial behaviour.

	<u>Anterior</u> <u>Group</u>	<u>Posterior</u> <u>Group</u>	<u>Control</u> <u>Group</u>	<u>p-value</u>
<b>Qualitative</b>				
Correct(pos)	0.7	1.4	0.6	0.38
Correct(neg)	0.1	1.3	0.4	0.11
Wrong(pos)	1.0	3.5	5.5	0.003
Wrong(neg)	5.8	9.6	8.0	0.2
<b>Self-Report</b>				
Positive	8.9	7.7	7.6	0.56
Negative	7.0	6.7	7.2	0.96

Table 17

Mean scores for the lesion and control groups on qualitative and self-report indices of emotional response.

**Self-report:** Subjects had to rate their reactions to the card sorting situation on 6 negative adjectives and 4 positive adjectives. Each adjective was individually rated on a 4 point scale. Ratings for all adjectives were then summed, giving a total emotional response score. If anterior damage produces a general flattening of all emotional responses, we would predict that the total

emotional response score would be significantly lower in the anterior lesion group. However the anterior and posterior groups did not differ on this measure [ $F(2,35)=0.36$ ;  $p=0.70$ ]. Alternatively, it has been suggested that positive mood is relatively preserved or enhanced in the anterior lesion group, and negative subjective response is reduced in the anterior group. To test this, ratings for the 4 positive adjectives were summed to give a positive subjective response score. Similarly, a negative subjective response score was derived from the sum of the ratings of the 6 negative adjectives. However the 3 groups did not differ on the composite measures of positive [ $F(2,35)=1.84$ ;  $p=0.17$ ], or negative [ $F(2,35)=0.03$ ;  $p=0.97$ ] subjective response.

There are at least two ways of accounting for the absence of an anterior-posterior difference on these self-report measures. First, equivalence of the lesion groups on the composite measures of subjective reaction might be explained by rejecting the hypotheses of (a) a relative suppression of negative emotional responses, of (b) a general suppression of emotional response in the anterior lesion group. Indeed, there are some grounds for taking this course. For example, human frontal damage has been associated with an increased incidence of negative emotional states such as depression (Robinson and Szelata, 1981), and negative emotional behaviours such as increased irritability (Lishman, 1978). Furthermore, in the rhesus monkey increased social anxiety has been reported after frontal lesions (Butter and Snyder, 1972). Therefore, perhaps some "negative" emotional reactions are enhanced in the anterior lesion group, while other "negative" reactions are reduced in intensity. Also, we cannot be confident that positive emotional experience is generally preserved or enhanced in the frontal group, especially as the euphoria displayed by these patients tends to be mild and passive rather than excited and exalted (Lishman, 1978; Hecaen, 1964).

	<u>Anterior</u> <u>Group</u>	<u>Posterior</u> <u>Group</u>	<u>Control</u> <u>Group</u>	<u>p</u>
Confuse	1.7	2.4	2.2	0.03
Frustrated	1.5	1.9	1.8	0.69
Irritated	1.2	1.3	1.7	0.69
Childish	1.0	0.2	0.3	0.03
Upsetting	0.4	0.0	0.8	0.02
Anxiety	0.9	0.9	1.1	0.85
Interesting	2.5	2.2	2.2	0.95
Enjoyable	1.7	2.0	2.0	0.49
Fun	2.1	1.9	1.4	0.48
Satisfying	2.0	1.7	1.8	0.85

Table 18

Mean rating for each adjective on self-report checklist assessing subjective reaction to card sorting situation. Kruskal-Wallis one-way ANOVA was used to assess significance.

Perhaps patients' subtle, and sometimes complex emotional responses to the card sorting situation cannot be meaningfully captured by the composite negative and positive subjective reaction scores. To seek a more accurate assessment of subjective emotional differences, the ratings for each adjective made by anterior, posterior and control patients were considered individually. Table 18 shows that the 3 groups differed significantly on three of the negative adjectives. Subsequent Mann-Whitney U tests indicated that the anterior group rated the task as significantly more "childish" and less "confusing" than the other two groups; while posteriors and controls did not differ in their ratings of these adjectives. Posterior patients reported themselves less "upset" than either the anterior or control groups; but the latter two groups did not differ significantly. These differences in self-report do not appear to permit a single clear-cut or straightforward interpretation, but frontal patients are reputed to have a loss of insight into their deficits, and this could account for their

reduced sense of confusion. On the other hand, one feature of the loss of insight is a tendency to rationalise deficits, and perhaps this could account for the tendency of the patients with anterior lesions to see the card sorting task as childish. Of course, these are post hoc speculations and, in any case, it is not clear why patients with posterior lesions should feel less upset than controls and anteriors.

There is a second way of accounting for inability to differentiate the anterior and posterior lesion groups on the **composite** positive and negative subjective reaction measures. Our difficulty in demonstrating a general reduction in emotional response or a selective reduction in negative subjective reaction might lead us to question the validity of the self-report measure. Certainly, there was some anecdotal support for this possibility. On a few occasions the investigator felt sure a patient experience little or no subjective reaction in the card sorting situation, yet the patient rated himself as experiencing significant positive and/or negative response. In other instances, the patient appeared more upset than their subjective negative reaction scores would indicate. Such discrepancies might be caused by weaknesses common to many self-report measures, including the effects of individual response styles and the tendency to choose socially desirable responses (Anastasi, 1969). Also, the findings might have been more illuminating had the adjective checklist been presented in a slightly different way. In the present study the patient was asked to give a general assessment of the card sorting task – they had to say whether, **overall**, it was confusing, fun to do etc. However patients sometimes complained that it was hard to make this general assessment because when they were sorting cards correctly the task was pleasant, while it was distressing when they experienced a sequence of incorrect card sortings. In retrospect the results may have been more illuminating and accurate if the

patients had been asked to give separate assessments of their subjective responses to being (a) wrong and/or (b) correct. The request that patients give their average reaction may have had the effect of eliminating the information of interest.

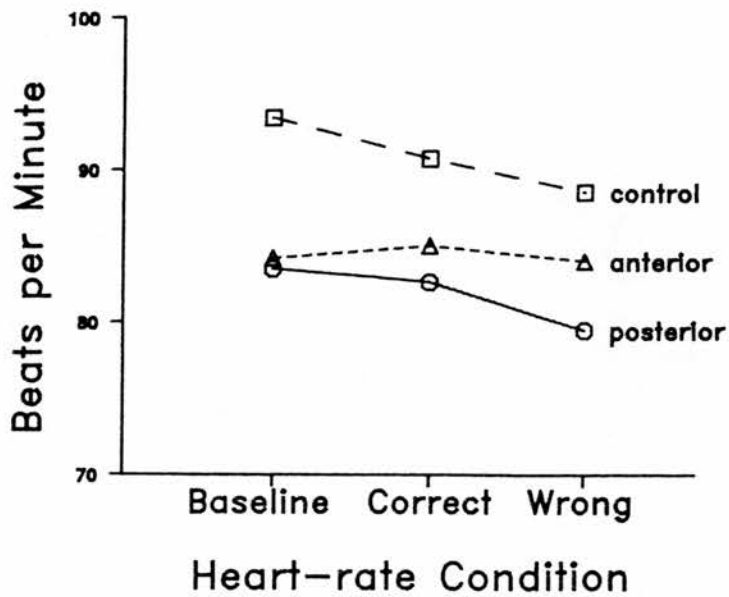


Figure 8

Heart-rate during baseline, correct, and incorrect card sortings in the three lesion groups.

**Autonomic indices:** To provide an index of the autonomic component of emotion the patient's heart rate was monitored throughout the video-recorded session. Baseline heart rate (B), average heart rate during the first 10 wrong card sorts (W), and average heart-rate during the first 10 correct card sortings (C) were monitored, in 9 anterior, 9 posterior, and 7 control subjects. In a

repeated measures analysis of variance, lesion group (anterior v posterior v control) was the between subjects factor, with repeated measures for heart-rate condition (B v W v C). If heart-rate provides a measure of the autonomic component of the patients' emotional response to the card sorting situation, we might predict that heart-rate during incorrect card sortings (W) would differ from heart-rate during correct card sortings (C), or baseline heart-rate (B). However though there is a trend in this direction (Figure 8), the effect of the within subjects heart-rate condition (B v W v C) fell short of significance [Wilks Lambda=0.8006; Approximate  $F(2,21)=2.62$ ;  $p<0.1$ ].

This weak association between heart-rate and correctness or incorrectness of card sorting might be explained in three ways. First, it may be that the patients' greatest emotional response was to the card sorting situation as a whole. For example, some patients may have viewed it as being a generally aversive task, so that they were equally anxious when they were correct as when they were incorrect. This view does not fit well with behavioural measures of facial expression, which showed patients' expression to be much more animated and emotionally charged after incorrect card sorts (Table 17). Also, patients often spontaneously observed that they much preferred the correct trials to the incorrect trials. Nevertheless it is possible that patients experienced a general pervasive emotional response to the test situation per se. We might therefore hypothesise that our heart-rate measures were more sensitive to this general emotional response than to fluctuations in emotion caused by correct and incorrect card sortings. Assuming this to be the case, if the frontal group suffered from a general reduction in emotional reactivity; we might predict that average heart-rate over all 3 conditions  $([B+W+C]/3)$  in the anterior group would differ from average level of heart-rate in the posteriors, or controls. However Figure 8 shows that this was not the case, and in the

repeated measures analysis of variance the effect of lesion group was not significant [ $F(2,22)=0.85$ ;  $p=0.45$ ].

A second way of accounting for the weak association between heart-rate and outcome of each card sort is to hypothesise that a "paradoxical" effect is occurring. For example, it may be that in one of the card sorting conditions heart-rate increases in the anterior lesion group, but it decreases in the posteriors and controls. Figure 8 raises the possibility that heart-rate after a wrong card sort falls in the posteriors and controls, but is unchanged throughout all card sorting conditions in the anterior group. To assess this statistically we would need to demonstrate a significant interaction between lesion group and heart rate condition, but this was not found [Wilks Lambda=0.5952; Approximate  $F(4,42)=0.65$ ;  $p=0.7$ ].

There is a third explanation for the general lack of association between the heart-rate measures and card sorting conditions. The heart-rate measures used may not provide valid indices of autonomic responsiveness. It is possible that other heart-rate measures would have been more successful in distinguishing the lesion groups. Certainly, the heart-rate measures devised by Ekman *et al.* (1983) were significantly related to emotional experience. In their experimental paradigm, heart-rate was highest when the subject had to re-live a situation which had made them angry, whereas lowest heart-rate occurred when the subject re-lived an experience of disgust.

There is, however, a more fundamental doubt about the validity of our heart-rate measures. In his review Elliott (1974) concludes, "There are.....grounds for skepticism about **any** view of the motivational significance of heart-rate." Though Elliott agrees that heart-rate alters as a function of motivational state he suggests that it is the arousal and/or the

somatic-behavioural component of the emotional response that is related to heart-rate. When an emotional response occurs in the absence of arousal or a behavioural response, heart-rate may not change. While planning the present study, it was concluded that GSR would have been a better measure of autonomic reactivity. Unfortunately, particular care is required when monitoring GSR, and time constraints prevented the inclusion of this autonomic index. It was felt highly desirable to at least attempt to measure autonomic reactivity, so heart-rate was chosen because it is easy to measure.

### Conclusions

1. Patients with posterior lesions and control patients showed no differences on any of the measures of spontaneous reaction to the failure situation.
2. Patients with anterior lesions showed a marked poverty of facial expression. Not only were reactions less frequent, but also reactions were less complex and contained fewer motor components.
3. The data indicated that poverty of facial expression was associated with lesions situated anteriorly, as well as in the basal ganglia. This effect of lesion site remained after partially controlling for lesion size, type and degree of bilateral damage.
4. Reduced facial expression occurred in the context of a general facial motor impairment, however it seemed that the flattened facial expression could not be entirely attributed to a primary motor deficit.
5. The reduced response to failure could not be wholly accounted for in terms of a cognitive/attentional deficit.
6. Multiple measures of emotional reaction were included to seek positive evidence that the reduced facial response in the card sorting situation is an emotional deficit. There was some suggestion of a frontal deficit on the qualitative ratings of emotional reaction.
7. Limited support for an anterior-posterior difference in emotional response was found using the self-report measure.



8. Heart-rate indices did not distinguish the anterior and posterior lesion groups. They did not correlate with facial expression of feeling.

## 5.2. Test of Voluntary Production of Emotional Expression

Subjects were asked to make a disgusted, a surprised, an angry, and a smiling face. The measures derived were:

- Total number of FACS action units produced for the 4 facial expressions.
- The voluntary expressions were given a qualitative rating indicating the degree to which the subject accurately portrayed the 4 emotions.

<u>Action Units</u>	<u>Anterior Group</u>	<u>Posterior Group</u>	<u>Control Group</u>
18-17			1
16-15		4	
14-13	4	5	3
12-11	2	4	4
10-9	3	1	1
8-7	2		
6-5	2		
4-3	1		

Table 19

Total number of FACS action units produced during the test of voluntary expressed emotion in the anterior, posterior and control groups

In a 3x2 analysis of variance total number of action units produced in the voluntary production of facial emotional expression was the dependent variable,

with lesion group (anterior v posterior v control) and sex as factors. The effect of the lesion group factor was significant [ $F(2,32)=8.13$ ;  $p<0.001$ ]. Neither sex [ $F(1,32)=1.29$ ;  $p=0.27$ ] nor the sex by site interaction [ $F(2,32)=1.08$ ;  $p=0.36$ ] achieved significance. Therefore in subsequent analyses the 21 male and the 17 female subjects are grouped together, and Table 19 shows the distribution of total FACS scores for the three groups.

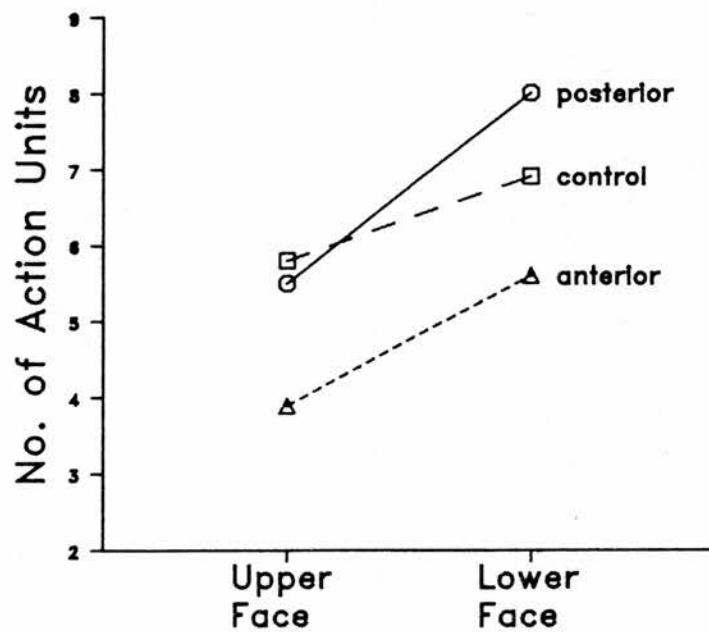


Figure 9

Upper and lower face movement in test of voluntary emotional expression in the anterior, posterior and control groups.

A one-way analysis of variance was performed, where controls ( $N=9$ ), anterior ( $N=14$ ) and posterior ( $N=14$ ) patients were compared, and the

dependent variable was total FACS action units produced in the voluntary production of emotional expression. Post hoc Scheffe tests showed that the anterior group made significantly fewer FACS action units than controls or posteriors, whereas controls and posteriors did not differ.

As cortico-bulbar projections to the nucleus of the 7th nerve are bilateral for the upper face and unilateral for the lower face a 3x2 repeated measures analysis of variance was performed with face (upper v lower) as the within subjects factor, and lesion group (anterior v posterior v control) as the between subjects factor. More lower face movements were made in emotional expression by all groups [ $F(1,35)=19.81$ ;  $p<0.0001$ ]. Of greater interest is the face by group interaction, which fell far short of significance [ $F(1,35)=0.86$ ;  $p=0.43$ ]. Figure 9 confirms that upper and lower face movement are both more or less equally reduced in the anterior group.

#### Voluntary emotional expression with lesion size and location

**Lesion size and aetiology:** Patients with frontal lobectomy were excluded and the influence of total lesion size was partialled out in a one-way analysis of covariance, with total action units as the dependent variable. Lesion size was not significantly related to voluntary emotional expression score [ $F(1,20)=1.26$ ;  $p=0.28$ ], however the anterior group remained impaired relative to posteriors [ $F(1,20)=16.94$ ;  $p<0.001$ ]. To control for lesion type as well as size patients with frontal lobectomy, or meningioma, or a metastasis, or stroke, or AVM excision were excluded and lesion size was again partialled out as a covariate. As before the lesion size covariate did not contribute significantly to voluntary emotional expression score [ $F(1,11)=0.024$ ;  $p=0.88$ ], and the 9 anterior patients showed significantly reduced voluntary facial emotional expression relative to the 5 posteriors [ $F(1,11)=5.22$ ;  $p=0.43$ ].

Patients with unilateral (N=6) and bilateral (N=8) anterior lesions did not differ significantly on this test (Mann-Whitney  $U=16.5$ ;  $p=0.35$ ). Furthermore, controlling for the greater degree of bilateral involvement in the anterior group, 6 patients with a unilateral and one bilaterally lesioned patient were compared with 15 patients who had a posterior lesion, where one posterior patient had a bilateral lesion. Again patients with anterior damage showed significant impairment ( $t=2.24$ ;  $p=0.036$ ).

**Lesion site:** Patients with (N=13) and without (N=27) medial temporal damage performed equally well on this task ( $t=0.82$ ;  $p=0.42$ ). Patients with (N=21) and without (N=18) damage to the basal ganglia were also compared, and significantly reduced voluntary emotion expression was found in patients whose lesions extended into the corpus striatum ( $t=2.79$ ;  $p=0.009$ ). However in this comparison, about half of the patients whose lesion did not extend into the basal ganglia had no other subcortical damage. Also, lesion size was larger in patients with striatal involvement. Therefore in a further analysis patients whose lesion involved only one subcortical structure, and who had not had a frontal lobectomy were considered. The 3 patients with damage to the basal ganglia were compared with the 5 patients with medial temporal, or thalamic involvement, but there was only a non-significant trend for reduced voluntary facial emotion in the striatal group (Mann-Whitney  $U=3$ ;  $p=0.13$ ).

#### Reduced voluntary expression as a primary motor deficit

Table 16 shows that patients with anterior lesions made fewer upper face movements in spontaneous speech. The anterior group also made significantly more errors than the posterior lesion patients when imitating complex facial movement sequences. This was taken to indicate that the patients with anterior damage suffered from a general disorder of facial movement. Though it was

suggested that the general facial-motor deficit could not adequately account for the reduced spontaneous facial expression in the anterior lesion group, it has been argued that spontaneous and voluntary emotional expressions are controlled by separable neural systems. It is therefore necessary to examine the possibility that the anterior impairment of voluntary emotional expression is attributable to a general facial-motor deficit.

Voluntary emotional expression scores of patients with anterior and posterior lesions were compared in a one-way analysis of covariance. The combined covariates, upper face movements during speech and errors in imitation of facial movement, were significantly related to voluntary emotional expression score [ $F(2,18)=9.08$ ;  $p=0.002$ ]. However after adjustments were made for this effect the anterior lesion group ( $N=9$ ) remained significantly impaired relative to the posteriors ( $N=13$ ) in voluntary emotional expression [ $F(1,18)=4.83$ ;  $p<0.05$ ].

#### Voluntary expression as a test of emotion

So far, data from the tests of voluntary and spontaneous emotional expression lead to similar conclusions. The anterior group is deficient on both tasks, and the anterior impairment cannot be easily attributed to the uncontrolled influence of lesion variables, or to a general facial-motor deficit. For the spontaneous emotional situation we also considered the possibility that reduced facial expression in the anterior group was due to a lack of awareness of error. It seems implausible to attribute the reduced voluntary emotional expression in the anterior patients to a lack of insight, as all patients appeared to understand the rather simple test instructions. In short, on the basis of negative evidence we might conclude that an emotional disorder is responsible for the deficit of voluntary emotional expression of the frontal patients.

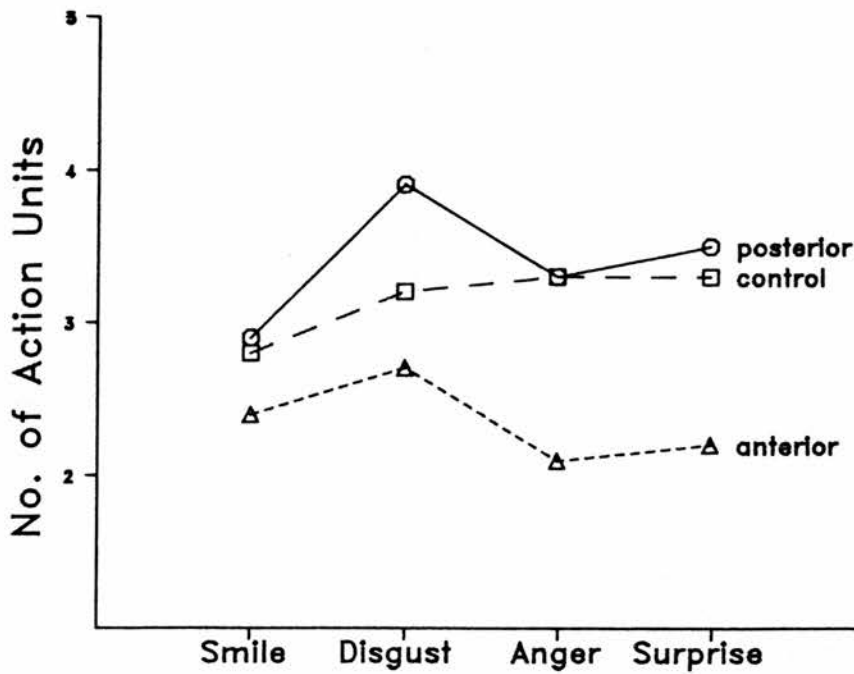


Figure 10

Mean action units produced by the anterior, posterior, and control patients in the voluntary production of happy, angry, disgusted and surprised faces.

However it has already been stressed that conclusions based on negative evidence, however suggestive, should be complemented by positive evidence where possible. Therefore in addition to counting the FACS action units in the voluntary expressions, each expression was given a qualitative rating of the adequacy of the attempt to portray a particular emotion. The anterior lesion group produced significantly less adequate emotional expression than the posteriors ( $t=5.05$ ;  $p<0.001$ ). Furthermore, number of FACS action units produced correlated significantly with the qualitative rating of adequacy of emotional expression ( $r=0.73$ ;  $p<0.001$ ). Of course, we must remember that these two measures of voluntary expression may not be independent, as both are taken from the same video sequence. It is therefore necessary to find stronger positive evidence.

Further positive support would be gained if we could demonstrate a selective impairment in the expression of a specific type of emotion. In particular, we have been assessing the hypothesis that positive emotion is relatively spared, or enhanced, after anterior damage. However Figure 10 shows the anterior group was generally impaired for all 4 emotions expressed. While there was a non-significant tendency to produce fewer action units when smiling voluntarily (Mann-Whitney  $U=69$ ;  $p=0.12$ ), the patients with anterior lesions produced significantly fewer action units for their angry (Mann-Whitney  $U=57$ ;  $p=0.37$ ), disgusted (Mann-Whitney  $U=53.5$ ;  $p=0.023$ ), and surprised (Mann-Whitney  $U=49.5$ ;  $p=0.012$ ) facial expressions.

### Conclusions

1. Patients with anterior lesions performed less adequately than those with posterior lesions on the test of voluntary production of emotional expression.
2. The greater anterior deficit on this task was not easily attributable to the lesion variables of size, type or degree of bilateral damage.
3. There were indications that damage to the basal ganglia may be particularly liable to impair voluntary emotional expression, but medial temporal structures were not especially implicated.
4. General facial-motor impairment did not seem to fully account for the deficit in the voluntary production of emotional expressions.
5. There was some weak positive evidence indicating that the deficit in voluntary expressions was specifically emotional. Qualitative ratings indicated that the anterior patients' attempts to portray the 4 emotions were significantly less convincing.
6. No support was gained for the hypothesis that expression of positive emotion was relatively spared in the anterior group.

### 5.3. Discussion

**Spontaneous v Voluntary Emotional Expression:** It has been suggested that voluntary and spontaneous expression of emotion are each controlled by different neural systems (Ekman *et al.*, 1981; Steklis and Raleigh, 1979). In the present study, however, the tests of voluntary emotional expression and of spontaneous emotional expression gave rather similar results. Compared with posteriors and controls, voluntary and spontaneous facial expression were both relatively impoverished in the anterior lesion group. Also, on both tests the anterior deficit was fairly generalised with upper face and lower face movement equally affected. Furthermore, spontaneous facial expression was significantly associated with damage to the basal ganglia, while the link between striatal damage and impaired voluntary emotional expression approached significance. In conclusion, these data do not provide strong support for the hypothesis that voluntary and spontaneous emotional expressions are regulated by two distinguishable neural systems. However they are not entirely incompatible with this possibility, as voluntary and spontaneous expression scores are non-significantly correlated (Table 20).

**Validity of emotional tests:** A central aim of the present study was to evaluate the validity of the emotional tests. At first sight it might appear that it was unnecessary to seek to establish that the tests of perception and expression are especially sensitive to emotional deficit. For example, one can reasonably argue that in the card sorting situation it is likely that many patients did not like to be told that they were wrong. Presumably their facial response reflected their negative reaction to some degree. Similarly, when patients were asked to re-live feelings of being happy or angry in the voluntary emotional expression test, it is likely that their facial behaviour was partially conditioned



by their felt emotion. When we find deficits on these expressive tests in the anterior lesion group we are inclined to infer that the reduced facial response is due to an emotional deficit, and it is always possible that some other factor is responsible for the deficit. Therefore in the present investigation the validity of the emotional measures was examined in some detail.

1. A coding system of established reliability (FACS) was employed to assess spontaneous and voluntary facial behaviour, and an acceptable level of interjudge reliability was achieved when rating spontaneous facial reactions ( $r=0.92$ ). Unfortunately it was not possible to assess interjudge reliability for the test of voluntary expression. This was particularly problematic because patients often made several attempts to produce the appropriate feeling, and the investigator's discretion was used in the selection of the 4 video sequences to be rated. Time constraints prevented the investigator from comparing his decisions about the subject's best attempts against the decisions of an independent judge. Nevertheless test-retest reliability was found to be high ( $r=0.95$  for quantitative rating, and  $r=0.86$  for qualitative rating). There was a four month delay between ratings and the investigator did not consciously recall the details of the initial rating at the re-test.

2. Test validity can be markedly influenced by the way in which the test is presented. For example, in the card sorting situation the patient was asked to give an overall assessment of their reactions to the task. A significant proportion of patients found this difficult and, in retrospect, it might have been more revealing to have asked the patients to describe their reactions to being told they were making (a) wrong, and then (b) correct card sorts. Clearly, before we can produce valid tests of emotion we must carefully consider the way we present tasks to the patient. We cannot rely on face validity and assume that a task is measuring emotional deficit because we are presenting apparently

emotional test stimuli or requesting apparently emotional responses.

3. No matter how reliable and appropriately presented an emotion test might be, the possibility remains that a deficit in test performance is due to a perceptuo-motor or cognitive impairment rather than to an emotional disorder. The need to rule out a perceptual/cognitive explanation for deficits in perception of facial emotional perception has received some attention (Etcoff, 1984; Dekosky *et al.*, 1980); and the possibility that the deficits in spontaneous facial expressions might be due to general facial-motor disorder has also been considered (Kolb and Milner, 1981b). In the present study it was concluded that the reduction of spontaneous and voluntary expression could not be easily attributed to a general facial-motor disorder, on the grounds that an anterior-posterior difference remained after partialling out scores on tests of non-emotional facial movement. However such statistical controls do not provide compelling evidence, as it remains possible that the reduced facial movement in the failure situation indicated a general motor deficit which was dissociable from the motor disorders underlying the reduction in upper face speech markers and the increased errors in imitating facial movement sequences. Certainly, deficits on these later two tasks were dissociable ( $r=-0.12$ ;  $df=30$ ;  $p=0.51$ ).

4. It is argued that positive evidence is necessary to establish a test as being especially sensitive to emotional deficit. Unfortunately the present study did not provide strong positive evidence. One condition for positive evidence would have been fulfilled if it had been shown that one type of emotional behaviour was selectively impaired after damage in a particular neural site. This criterion was not met. No support was found for the proposition that negative emotional reactions were selectively reduced after anterior cerebral damage.

An alternative strategy for gaining positive evidence of emotional deficit that might be considered relies on the use multiple measures of emotional response. Most of the recent work on emotion has employed only one type of emotional measure. Researchers have tended to separately study visual emotional perceptions (Cicone *et al.*, 1980), or auditory emotional perception (Heilman *et al.*, 1975), or spontaneous emotional expression (Buck and Duffy, 1980), or voluntary emotional expression (Bruyer, 1981). In the present investigation these 4 categories of test were administered to the same patient. The fact that the anterior lesion group was impaired on all 4 tests adds weight to the view that the frontal lobes are specialised for the regulation of emotion.

Use of multiple measures is probably not a totally satisfactory means of gaining the necessary positive evidence for the hypothesis that the frontal lobes are specialised for emotional functions. We have seen that voluntary and spontaneous emotional expression scores were not highly correlated, and in fact table 20 shows that the correlations between various emotional indices were often rather low. Of course, it is always possible that with more sensitive tests, the different emotional measures would intercorrelate more highly. There is however a more fundamental obstacle to our reliance on multiple emotional measures to provide positive evidence of emotional deficit. Multiple measures would be ideally suited to provide the necessary positive evidence if all the components of emotional response were normally tightly coupled and controlled by a single central neural cortical-subcortical-brainstem emotional system. However available data indicate that self-report, autonomic and behavioural components of emotion are not tightly coupled in normals. For example, in non-brain damaged subjects Morrow and Labrun (1978) found self-report anxiety measures were not significantly correlated with heart-rate physiological indices of anxiety. Furthermore, animal data also suggests that it

is possible that separate neural systems control distinguishable emotional functions (Flynn and Smith, 1980a&b).

	Vet	Spont	Vol	SrNeg	SrPos	HrW	HrCor
Fet	0.76**	0.24	0.36*	-0.12	-0.02	0.05	0.14
Vet		0.25	0.39*	-0.15	-0.04	0.24	0.33
Spont			0.25	0.24	-0.01	-0.08	-0.05
Vol				0.21	0.13	0.41*	0.44*
SrNeg					0.17	0.02	0.00
SrPos						-0.03	0.00
HrW							0.96**

Table 20

Intercorrelation between emotional measures within the brain damaged sample. Vet - Perception of verbally expressed emotion; Fet - Perception of facially expressed emotion; Spont - Spontaneous FACS action units in failure situation; Vol - FACS action units in voluntary emotional expression test; SrNeg - Composite subjective negative reaction to failure situation; SrPos - Composite subjective positive reaction to card sorting task; HrW - Heart-rate when making wrong card sorts; HrCor - Heart-rate when making correct card sorts. (\*\*- $p < 0.01$ ; \*- $p < 0.05$ )

Necessary procedures for the validation of tests of emotional deficit have yet to be specified in precise terms. There is a clear need to assess the validity of emotional tests and they also suggest that such investigations may lead to theoretically interesting conclusions.

## CHAPTER 6

### PERCEPTION OF EMOTION AND HEMISPHERIC ASYMMETRIES

The tests of perception of facially expressed and of verbally expressed emotion were constructed to investigate three hypotheses about hemispheric asymmetries and emotional perception. First, primary asymmetries in perceptual and cognitive functions may be partially, or wholly, responsible for observed hemispheric asymmetries in emotional perceptions. Secondly, the right hemisphere is dominant for the perception of emotion. Thirdly, the right hemisphere is specialised for the perception of negative emotion while perception of positive emotion is the particular province of the left hemisphere.

#### **6.1. The Test of Perception of Facially Expressed Emotion**

Total scores for the 10 control, 19 right hemisphere, and 17 left hemisphere patients are given in table 21. When these three lesion groups were compared in a one-way analysis of variance, the group effect was significant [ $F(2,43)=17.43$ ;  $p<0.0001$ ]. The Scheffe procedure was used to make post hoc comparisons of group means. Both right and left lesion groups were significantly impaired with respect to the controls. However the difference between the right and left hemisphere groups fell short of significance.

**Handedness:** Whereas 6 of the subjects in this comparison were not fully right handed, studies of hemispheric functional asymmetries commonly recruit only fully right handed subjects. This is because language functions are lateralised in the left hemisphere of almost all right handers. Nevertheless, 70% of non-right handers have language functions represented in the left hemisphere, and a further 15% of non-right handers have bilateral language

functions (Passingham, 1979). Unfortunately rather Less is known about the lateralisation of visuo-spatial-perceptual functions in left handers, but after assessing language and visuo-spatial-perceptual capacities in 73 left handed patients with unilateral lesions, Hecaen and Sauguet (1971) concluded "...the functional hemispheric asymmetry found in right-handed subjects is also present in left-handed subjects, though to a less strong and consistent degree."

<u>Score Range</u>	<u>Right Hemisphere</u>	<u>Left Hemisphere</u>	<u>Controls</u>
40-37	1		1
36-33		1	5
32-29		3	4
28-25	2	4	
24-21	5	3	
20-17	3	4	
16-13	4	1	
12-9	4	1	

Table 21

Total scores on the test of facially expressed emotion for the control, right and left hemisphere groups

Given these uncertainties and deviations in organisation of cerebral function in non-right handers, it was necessary to compare total scores for the 3 lesion groups after excluding the 1 right hemisphere and 5 left hemisphere non-right handed patients. The group effect remained significant [ $F(2,38)=16$ ;  $p<0.0001$ ]; post hoc tests again showed the right and left hemisphere groups were impaired relative to the controls; while the difference between the right and left lesion groups again fell short of significance.

Two analyses were performed for all subsequent comparisons of right and left hemisphere groups discussed in Chapters 6 and 7. In the first analysis all patients are included, while the second analysis considers only right handed patients. These two analyses always produced a similar result, except that significance levels tended to be higher when all subjects were used in the comparisons. Therefore only the result of the analysis using all patients will be reported in this chapter and the following chapter.

**Sex differences:** No sex differences in emotion test scores have been found, in data discussed in the previous two chapters. However the inter-hemispheric dimension was not considered, and some have hypothesised that cerebral functions are differently lateralised in the male and female brain (Inglis *et al.*, 1982; McGlone, 1977). To test this possibility total facial emotional perception scores was the dependent variable in a 2x2 analysis of variance, with hemisphere (right v left) and sex as factors. If emotional functions are differently lateralised in the male and female brain it was predicted that the hemisphere by sex interaction would be significant. In the event, the sex by hemisphere interaction did not achieve significance [ $F(1,27)=1.94$ ;  $p=0.18$ ], and as before total mean scores for males and females did not differ significantly [ $F(1,27)=0.1$ ;  $p=0.76$ ]. Therefore in subsequent analyses the 25 males and the 22 females will be treated as identical.

#### Perception of facially expressed emotion with lesion size, type and location

**Lesion size and aetiology:** Mean total lesion size was slightly larger in the right hemisphere group (88.3cc), compared with the left hemisphere group (80.6cc), but this difference fell far short of significance ( $t=0.57$ ;  $p=0.57$ ). Also, table 3 shows that the hemispheric groups were roughly comparable in terms of aetiology except that all 3 metastases were in the right hemisphere and all 3

arteriovenous malformations were in the left hemisphere. To control for the possible confounding influences of lesion size and lesion type, total facial emotional perception scores of the right and left hemispheric groups were compared in an analysis of covariance. Patients with metastasis or AVM were excluded and lesion size was partialled out as the covariate. The effect of the lesion size covariate did not achieve significance [ $F(1,28)=2.14$ ;  $p=0.15$ ], and the non-significant trend for greater deficit in the 16 patients with right hemisphere lesions remained [ $F(1,28)=2.87$ ;  $p=0.10$ ].

**Lesion location:** In the left hemisphere group 4 patients had anterior lesions and 7 patients had posterior lesions. Within the right hemispheric group, 3 of the lesions were anterior and 9 were posterior. Figure 3 shows composite lesion maps for these 4 subgroups, and mean scores for the 4 brain damaged subgroups and controls are represented in figure 11. As there were only 3 right anterior and 4 left anterior patients it was unclear whether the data met the assumptions necessary for the application of parametric statistics. Nevertheless, the evaluation of the relative performance of the 4 lesion subgroups is of some importance, therefore total perception of facially expressed emotion scores of the 4 lesion subgroups and controls were compared in a one-way analysis of variance.

The group effect was highly significant [ $F(4,28)=13.30$ ;  $p<0.0001$ ]. Post hoc testing using the Newman-Keuls procedure showed that control scores were significantly higher than scores in all 4 lesion groups. Also, the left posteriors performed at a significantly superior level relative to the right anterior, and right posterior, and left anterior subgroups. No other paired group comparisons were significant.



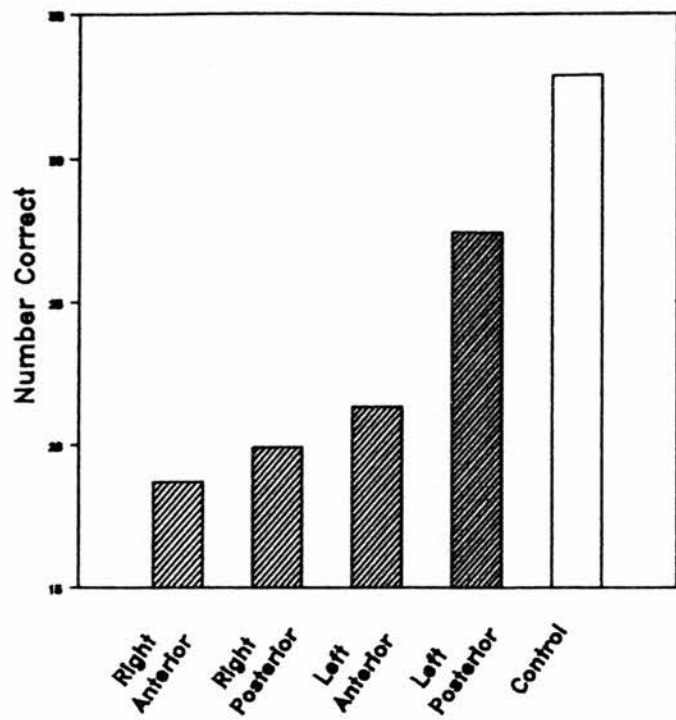


Figure 11

Total perception of facially expressed emotion scores in right anterior, right posterior, left anterior, left posterior, and control groups.

To account for the differential preservation of facial emotional perception in the 4 lesion groups, two statistical models will be considered - the model where main effects are additive, and the model where main effects are not additive. On the additive model, group performance is determined by the arithmetical sum of the inter-hemispheric main effect and the intra-hemispheric main effect. The intra-hemispheric main effect quantifies the relative performance of anterior and posterior lesion groups. It measures the extent to which mean facial emotion scores are depressed in the anterior group relative to the posteriors. On the other hand, the inter-hemispheric main effect quantifies the relative performance of the right and left hemisphere lesion groups. For illustrative purposes we will assume a significant

inter-hemispheric main effect where mean facial emotional perception scores are significantly depressed by a defined amount in the right hemisphere group, as compared with the left hemisphere group. Adding together this assumed inter-hemispheric effect with the known intra-hemispheric effect, greatest deficit is predicted in the right anterior group. Conversely, this additive model would predict least impairment on emotional test performance after left posterior damage. Finally, left anterior and right posterior lesions would result in intermediate degrees of deficit. Indeed, figure 11 shows that the magnitude of mean scores in the 4 brain damaged groups follows this relative order.

In the non-additive statistical model there is an interaction between the inter-hemispheric and intra-hemispheric factors. In this case, mean scores of the 4 groups would significantly depart from the relative magnitudes predicted by the additive model. There are several ways in which inter- and intra-hemispheric factors might interact. One possible departure from the additive model would be enhanced deficit in one or more of the brain damaged groups. For example, if the right hemisphere and the frontal lobes are each specialised for different emotional functions, then unique conditions for emotional control might be found at the interface between the anterior and right hemisphere emotional neural systems. The combination of the two emotional systems meeting in the right frontal lobe might create the conditions for certain new, additional or emergent emotional functions. If the two systems interact in this way, it follows that right anterior damage would disrupt these emergent functions, and consequently produce greater impairment on emotional tests than would be expected on the additive model.

The explanatory power of the additive and interactive models was compared in a 2x2 analysis of variance, with total facial emotional perception scores as the dependent variable; while the inter-hemispheric and intra-hemispheric

dimensions were factors. The additive model gave the better fit to the data, as the interaction between the inter- and intra-hemispheric factors was not significant [ $F(1,19)=1.27$ ;  $p=0.28$ ]. The inter-hemispheric factor was significant, with greater impairment in the right hemisphere group [ $F(1,19)=8.89$ ;  $p=0.008$ ]. The intra-hemispheric factor approached significance, with greater deficit after anterior damage [ $F(1,19)=3.07$ ;  $p=0.096$ ].

The 2x2 analysis of variance using the 4 smaller lesion subgroups gives similar results to the analysis of the inter- and intra-hemispheric effects with larger groups. In one of the latter main analyses all anterior and posterior patients were compared and significant impairment was found in the anterior lesion group. However there was only a trend for greater anterior deficit when the 4 lesion subgroups were considered. Furthermore, in the main analysis where all right and left hemisphere patients were contrasted, the right hemisphere group only tended to show greater deficit in total perception of facially expressed emotion scores. However the inter-hemispheric factor is significant using the 4 lesion subgroups. Small sample size is the most probable explanation for the fluctuations in significance levels between the main one-way analyses of variance and the analysis using the 4 lesion subgroups.

Small sample size also raises a serious doubt about the adequacy of our test of the significance of the interaction of the inter- and intra-hemispheric factors. However, despite small subgroup sizes, it was felt desirable to make a provisional assessment of the strength of the interaction between the inter- and intra-hemispheric factors, because, with a few exceptions (e.g. Pribram, 1981), the possibility of interaction between these two factors is not discussed. As researchers have tended to focus on either the inter-hemispheric or the

intra-hemispheric factor, they do not test the possibility of interaction, and so assume an additive statistical model by default.

**Impaired facial emotional perception as a secondary manifestation of perceptual/cognitive deficits**

	<u>Right</u> <u>Hemisphere</u>	<u>Left</u> <u>Hemisphere</u>	<u>t-value</u>	<u>p</u>
<b>Face Emotion</b>				
<b>Total score</b>	19.2	23.1	1.83	0.076
Face-face match	8.7	11.6	2.43	0.02
Face-word match	10.5	11.3	0.65	0.52
<b>Face identity recognition</b>	12.9	17.1	–	0.04
<b>Recognition memory</b>				
Faces	13.3	19.1	3.36	0.003
Words	17.3	15.8	0.73	0.47
<b>Intellectual tests</b>				
Verbal IQ	95.3	76.1	3.85	0.001
Performance IQ	83.3	94.2	2.22	0.036

Table 22

Performance of left and right hemisphere lesion groups on the test of perception of facial emotion, and on tests of general perceptual and cognitive functions. Where t-values are not given the non-parametric Mann-Whitney U test was used to estimate significance level.

Table 22 shows perception of facial emotion total scores tended to be lower in the right hemisphere lesion group. The right hemisphere deficit did not occur "across the board". These patients were selectively impaired when matching emotional faces to standard response faces, but the hemispheric

groups performed at a similar level when matching faces to standard names. However right lesioned patients were also inferior to the left hemisphere group for facial identity recognition, and facial recognition memory, and performance IQ. Conversely, Verbal IQ was significantly higher in the right hemisphere group. Thus the right hemisphere group had particular difficulty with visual perceptual/cognitive tests, and the left hemisphere groups had relatively more deficit on a verbal-cognitive measure.

	RIGHT HEMISPHERE			LEFT HEMISPHERE	
	Face-Face	Face-Word		Face-Face	Face-Word
Performance IQ	0.73***	0.39		-0.21	0.36
Verbal IQ	0.43	-0.02		-0.04	0.72***
Face Identity Recognition	0.76**	0.55		-	-
Recognition Memory					
Faces	0.38	0.49*		-0.01	0.02
Words	0.40	0.63**		0.00	0.04
Visual Neglect					
Line Cancellation	-0.62**	-0.28		-	-
Line Bisection	-0.56*	-0.33		-	-
Token Test	-	-		0.04	0.67**

Table 23

Correlations between general sensori-motor-cognitive measures and emotional face-face matching, and emotional face-word matching within the hemispheric groups. Face-face matching is particularly closely related with measures of visual-perceptual-cognitive deficit in the right hemisphere group. Face-word matching is significantly related to verbal-cognitive measures in the left hemisphere group. (\*\*\*-p<0.001, \*\*-p<0.01, \*-p<0.05)

**Right hemisphere perceptual-cognitive deficit:** Eight patients had clinically detectable visual perceptual disturbances, and in 6 of these a left visual field defect was found during the neurological assessment. In one case a bilateral lesion was largely confined to the right hemisphere, but the remaining 7 patients had only unilateral right hemisphere damage. Seven of the patients with visual perceptual disturbance completed the test of perception of facially expressed emotion, and were compared with the 10 other right lesion patients who showed no signs of visual perceptual deficit or visual field defect (one patient with equivocal evidence of visual disturbance was excluded). A strong association between visual perceptual deficit and face-face matching for emotional expression was found ( $U=6$ ;  $p=0.02$ ), but there was only a trend for greater deficit of emotional face-word matching in the patients with visual perceptual disturbance ( $U=19.5$ ;  $p=0.09$ ). Furthermore, table 23 shows that, after right hemisphere lesions, face-face emotional matching scores correlated significantly with Performance IQ, face identity recognition, and 2 measures of left visual neglect (line cancellation and line bisection). Presumably, face-face matching relies more heavily on visual perceptual skills than the face-word matching condition, which did not significantly correlate with the measures of visual perceptual/cognitive function (Table 23).

The lack of a significant association between face-recognition memory and emotional face-face matching appears to conflict with the conclusion that deficit in facial emotional perception in the right hemisphere group is secondary to visual perceptual/cognitive deficit. However there is evidence that memory and visual perceptual losses are dissociable. In particular, defects of facial memory can occur which are independent of perception (Milner, 1968; Warrington and James, 1967). Conversely, Whitley and Warrington (1977) describe a patient who demonstrated perceptual difficulties while matching

unknown faces, but facial recognition memory appeared relatively spared.

**Left hemisphere verbal-cognitive deficit:** Different cognitive deficits are associated with impaired perception of facially expressed emotion in the left hemisphere group. 10 patients had a marked disorder of oral expression or scored below the recommended cut-off of 29/36 on the short Token Test (De Renzi and Faglioni, 1978), and were classified dysphasic, with mean Token Test score of 17.8 (SD=7.5). In one case the lesion was largely confined to the left hemisphere, but the remaining 9 had only unilateral damage. These dysphasic and the 8 non-dysphasic left hemisphere patients performed equally well on the emotional face-face matching condition ( $U=37$ ;  $p=0.83$ ). However when required to name the emotion in the emotional face-word condition, the dysphasic group were significantly impaired ( $U=11.5$ ;  $p=0.009$ ). Similarly, face-face matching performance was unrelated to any measure of perceptual/cognitive function in the left hemisphere group (Table 23). On the other hand, score on the emotional face-word matching condition was significantly related to Verbal IQ and Token Test score (Table 23). As verbal-cognitive deficit is related to the condition where a verbal-naming response is required, we can conclude that deficit in facial emotional perception is, partly, secondary to a verbal-cognitive deficit following damage to the left hemisphere.

It might be argued that language and perception of facial emotion are distinct functional capacities which are subserved by anatomically adjacent cortical regions. Therefore with the large lesions incurred by the present patient population both functions are coincidentally impaired by the same lesion, and therefore a significant correlation between Token Test scores and perception of emotion scores is to be expected. A similar claim might be made for the association between visual perceptual deficit and face emotion

perception after right hemisphere damage. However, in the left hemisphere verbal/cognitive deficit correlated with face-word matching, while in the right hemisphere visual perceptual deficit correlated with face-face matching. This selective pattern of association suggests that impaired performance on the test of facial emotional perception is caused by different perceptual/cognitive deficits in the right and left hemispheric groups. If these correlations were purely coincidental we might have expected face-face matching and face-word matching conditions to **both** correlate significantly with the cognitive deficits characteristic of each hemisphere.

#### Right hemisphere dominance for emotion and the test of perception of facially expressed emotion

**Dissociation of emotional from cognitive deficit:** It has been concluded that a perceptual/cognitive deficit causes impairment on the perception of facial emotion test. However it remains possible that in right hemisphere disease facial emotional perception is impaired by a combination of (a) perceptual/cognitive deficit, and (b) disturbance of a lateralised emotional function. Two strategies were incorporated into the experimental design with the aim of dissociating the emotional from the perceptual/cognitive effects. The first approach was to manipulate the response requirements of the test of perception of facially expressed emotion. The face-word matching condition was of particular interest, as the perceptual/cognitive explanation would be supported if the left hemisphere group had more difficulty than right hemisphere patients when a verbal matching response was required. Conversely, the hypothesis of right hemisphere dominance for emotion predicts that greatest impairment of face-word matching would follow right hemisphere damage. In the event, both groups performed equivalently (Table 22).



Though problematic for the theory of right hemisphere emotional dominance, this does not force a rejection of the right hemisphere emotional dominance hypothesis. Both hemispheric groups were impaired relative to controls. It remains possible that deficit on the face-word matching condition is due to the combined effect of perceptual and emotional deficits in the right hemisphere group, while the left hemisphere patients' face-word matching deficit is due to the effect of a verbal-cognitive disorder. If, in statistical terms, the left and total right hemispheric effects happen to be of equal magnitude, there would be no right-left difference in total emotional face-word matching scores.

The second experimental strategy designed to dissociate asymmetrically lateralised perceptual and emotional functions involved analysis of covariance. It is assumed that scores on the perception of facial emotion test in the right and left hemispheres are determined by (a) a specific facial perceptual deficit measured by face identity recognition test, and (b) a specifically emotional deficit. Then, using the linear regression between face emotional perception and face identity perception scores, right and left hemisphere patients' facial emotional perception scores are adjusted to hold degree of facial perceptual deficit constant. If the adjusted facial emotional perception scores were still significantly lower in the right hemisphere group, we would have supported the hypothesis that a special right hemispheric emotional deficit is superimposed on the facial identity perception deficit. Indeed, Dekosky *et al.* (1980) found right lesioned patients had particular difficulty with an emotional face-face matching task, but after adjusting emotional face-face matching scores of each patient according to their degree of facial identity recognition deficit, the effect of side of lesion was no longer significant. The same result was found in the present investigation when a similar analysis of covariance was performed.

However, in both studies the relationship between dependent variable and covariate precluded use of analysis of covariance.

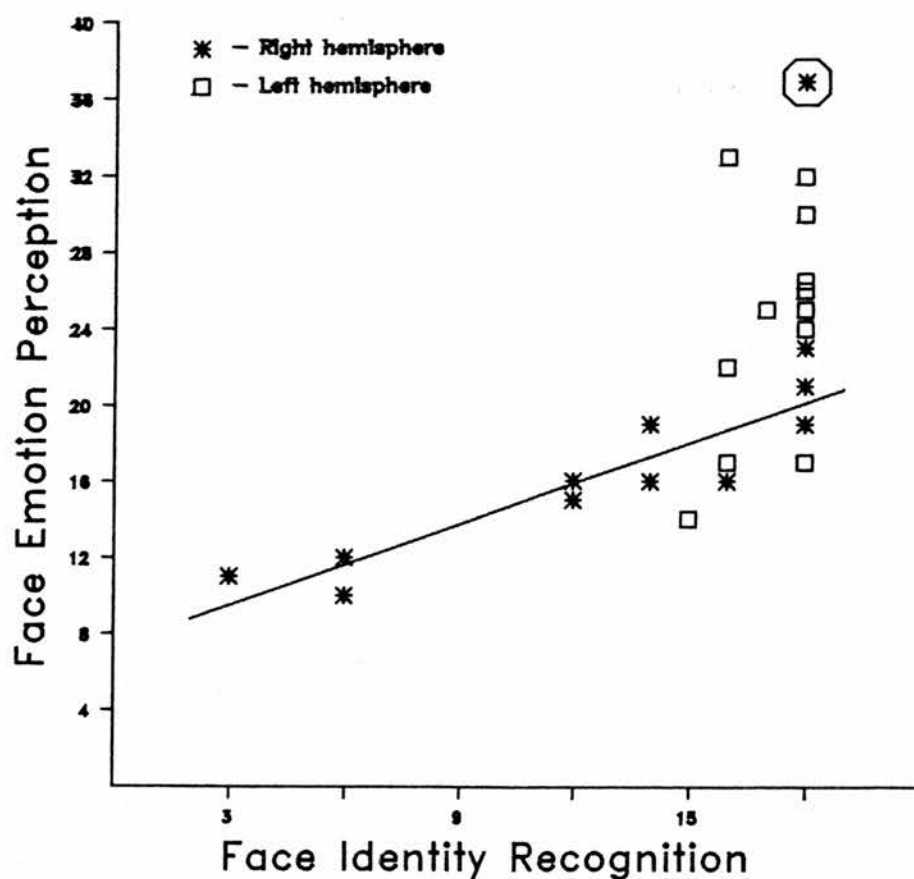


Figure 12

Relationship between face identity recognition scores and face emotion perception scores in the right and left hemisphere groups. The regression line between dependent variable and covariate fits right hemisphere data, except for one outlier (which is encircled). No meaningful regression line could be computed for the left hemisphere data.

Figure 12 shows that a linear regression equation could be computed for the present right hemisphere group, but not for the left lesioned patients

because only 4 of the left hemisphere patients made 2 or more errors in facial identity recognition. This problem was more serious for Dekosky *et al.* (1980) as it appears that none of their left hemisphere patients made an error on their prosopagnosia screening test. Presumably, face identity recognition tests used in both studies were too easy for the left lesioned patients. With a more sensitive face recognition test left hemisphere patients would make more errors, and a regression equation between facial emotional perception and patients' facial identity recognition could be constructed. However, in this case the regression equations between covariate and dependent variable would be computed for different ranges of scores in the two hemispheric groups, while analysis of covariance assumes a linear relationship over a common range of scores. Of course, we might simply extrapolate a constant linear relationship over the common range of scores for both regressions. However our difficulties in generating comparable regression equations may indicate a more fundamental violation of the assumptions on which analysis of covariance is based. The causal link between dependent variable and the new covariate may be quite different in the two hemispheric groups.

Let us accept, for illustrative purposes, that the linear relationship between face identity and face emotion recognition stems from a common underlying specific facial perceptual deficit in the right hemisphere group. We shall also make the more questionable assumption that in the left hemisphere group the link between dependent variable and covariate is mediated by a "general" verbal-cognitive deficit. In the analysis of covariance, face emotion scores would be adjusted for facial perception deficit in the right hemisphere group, and for general verbal-cognitive deficit in the left hemisphere group. Under these circumstances it would be difficult to interpret a significant right hemispheric deficit in adjusted scores. We could not safely infer that this

"residual" right hemispheric deficit indicated this hemisphere was specialised for emotional perception.

It is concluded that analysis of covariance was not a legitimate procedure with the present data. Nevertheless, it will be appropriate and desirable to apply this statistical technique in future work provided (a) a more sensitive test of facial identity recognition is used, and (b) on theoretical and experimental grounds it is reasonable to suppose that the relationship between dependent variable and covariate is mediated by the same (or similar) causal agencies in the right and left hemispheric groups.

Unfortunately at present the latter prerequisite for analysis of covariance cannot be fulfilled in a completely satisfactory manner. While the existence of hemispheric functional asymmetries is not in dispute, their causal basis is a subject of considerable controversy (Bradshaw and Nettleton, 1981; Marshall, 1981). In particular, there is no consensus about the nature of the relationship between emotional and perceptual/cognitive processes within the two hemispheres. However several models appear to agree, at least, that there is a close relationship between right hemispheric perceptual/cognitive processes and emotional processes. They suggest that the emotional disturbances following right hemisphere damage cannot be categorised as purely emotional disorders (with no cognitive aspect), nor are they merely secondary manifestations of general sensori-motor disturbance. In an important sense these right hemispheric disturbances are irreducibly cognitive-emotional. As the cognitive and emotional disturbances are different aspects of underlying cognitive-emotional disorder, it is impossible, in principle, to sharply dissociate emotional and all perceptual/cognitive deficits after right hemisphere disease. We must next assess the extent to which the present evidence supports these current theories.

**Right hemisphere dominance for attentional/emotional processes:** Heilman and his colleagues consider neglect and emotional indifference are two manifestations of the same underlying attentional/emotional deficit (Heilman *et al.*, 1978). Clearly, if an animal is to survive, attentional and emotional/motivational processes must interface. The animal's attention must be directed in relation to motivationally/emotionally important objects or agents. Watson *et al.* (1981) and Mesulam (1981) review animal and human data supporting this link between emotional and attentional processes. They suggest that spatial attention is directed by an interconnected neural network including frontal and parietal cortical fields, the cingulate gyrus, and the reticular formation. Though lesions in any component of this neural network can induce neglect, Mesulam (1981) and Watson *et al.* (1981) argue that each region makes a special contribution to the process. For example, it is hypothesised that the cingulate gyrus is particularly important for the integration of emotional and attentional processes. As well as being embedded within the neural network for the direction of spatial attention, the cingulate gyrus has strong anatomical links with the amygdala, hippocampus, hypothalamus and other limbic structures (Brodal, 1981; Baleydier and Maugiere, 1980). While the cingulate gyrus might be specialised in this way, Mesulam (1981) stresses that the neural network for the direction of spatial attention works as a whole. Therefore damage in any part of the network will result in motivational deficit. Thus Mesulam argues that damage in parietal or frontal cortex induces the motivational as well as sensory and motor components of the neglect syndrome. Of course, in man neglect symptoms are particularly common after right hemisphere damage. This further suggests that the neural network for attentional/motivational integrations is lateralised (to a significant but undetermined degree) in the right hemisphere (Heilman and Van Den Abell,

1980 & 1979; Heilman *et al.*, 1978).

If visual neglect and emotional dysfunction are manifestations of an attentional/emotional disorder, it would be predicted that line cancellation and line bisection scores would correlate significantly with capacity to perceive facially expressed emotion. This was found for face-face matching and neglect measures but, contrary to prediction, the association between face-word matching and neglect was much weaker and non-significant (Table 23).

This partial confirmation of the attentional/emotional hypothesis may be a consequence of limitations to the line bisection and line cancellation tests as measures of visual neglect. From clinical observations of patient behaviour during testing, only 6 patients showed clear indications of left sided neglect. Two other patients showed marked visual-perceptual disturbances, but gave no indication of left sided neglect in their test behaviour. Both patients performed normally on the line bisection task. Though the patient with a left visual field defect failed to cross out half of the lines in the cancellation test, there was no indication of neglect as lines were missed equally in the right and left sides of the page. To more accurately assess the attentional/emotional hypothesis the 5 patients with unequivocal neglect who had also completed the test of perception of facially expressed emotion were compared with the remaining right hemisphere patients (including the 2 patients with visual perceptual deficit but no neglect). The group with neglect were significantly impaired for emotional face-face matching ( $U=4.5$ ;  $p=0.003$ ), and tended to suffer greater deficit for emotional face-word matching ( $U=14$ ;  $p=0.075$ ).

In man neglect phenomena are associated with anterior and posterior right hemisphere lesions (Heilman and Valenstein, 1981; Damasio *et al.*, 1980; Chedru *et al.*, 1973; Colombo *et al.*, 1978). However figure 11 shows that facial

emotional perception was also impaired in the left anterior lesion group. This is not necessarily incompatible with the view that attentional/emotional processes are asymmetrically represented in the right hemisphere, and certainly Valenstein and Heilman (1979) advocate a special role for the frontal lobes in the regulation of emotion. It is possible that attentional/emotional processes are lateralised in the right hemisphere, whereas certain distinct (but as yet undefined) emotional functions are bilaterally represented in frontal and other cortical-subcortical limbic pathways.

**Differential hemispheric functional organisation:** Heilman, Watson, Mesulam and colleagues do not appear to commit themselves to a reason why the attentional/motivational function is lateralised in the right hemisphere. It may be a purely accidental quirk of nature that certain facial perceptual skills and attentional/motivational mechanisms both happen to be located in the right hemisphere, rather than the left, hemisphere. It may similarly be coincidental that language and certain voluntary movement skills are both represented in the left hemisphere. However many consider there are fundamental communalities between the range of deficits seen after right hemisphere disease, and after left hemisphere damage. These theories postulate a basic difference between right hemisphere and left hemisphere processes. Thus Hecaen and Angelergues (1963) suggested that in the left hemisphere different functions are represented in comparatively discrete cortical zones. They suggested that the focal organisation and language skills made the left hemisphere specialised for conceptual-analytic functions. In contrast, functions were thought to be represented in large equipotential zones of the right hemisphere. This gives the right hemisphere a special facility for making multimodal sensory integrations. Gainotti (1972) uses this view to account for the sensory neglect syndrome after right hemisphere damage. He argues that



neglect is a consequence of disturbance of the capacity to make multimodal sensory integrations, and he further suggests that these multimodal sensory integrations confer an emotional aspect to sensory experience. Thus damage to the right hemisphere simultaneously causes perceptual deficit and emotional disturbance. Semmes (1968) developed a slightly different model for hemispheric processing asymmetries but nevertheless similarly concluded that functions were more focally represented in the left hemisphere, and more diffusely represented in the right hemisphere.

Figure 11 offers limited support for this hypothesis. On one hand, within the supposedly "diffusely" organised right hemisphere **both** anterior and posterior damage result in equal impairment of facially expressed emotion. On the other hand, within the left hemisphere functions did seem to be more regionally organised, as anterior lesions appeared to cause greater deficit than posterior lesions, although this difference between left anterior and left posterior subgroups was not significant ( $U=5$ ;  $p=0.17$ ). A further prediction of the focal/diffuse hypothesis is that intercorrelations between test scores should be higher in the diffusely organised "equipotential" right hemisphere, as compared with the focally organised left hemisphere. Table 23 gives some support for this, because in the right lesion group 6 out of 14 correlation coefficients are significant, whereas in the left hemisphere group only 2 out of 10 coefficients achieved significance. Furthermore, the non-significant correlation coefficients are generally rather lower in the left hemisphere group.

**Emotional integrations of the right hemisphere:** Winner and Gardner (1977) and Gardner *et al.* (1975) suggest a link between visual-spatial, emotional, and certain linguistic deficits associated with right hemisphere disease. They do not make it entirely clear whether they consider visual-spatial, emotional and some linguistic functions as separate right hemisphere processes. However at the



very least these capacities appear to share certain formal or structural similarities. For example, right brain damaged patients can have difficulties in integrating visual objects within a spatial framework. Such patients can suffer spatial disorientation where they tend to get lost when traversing familiar routes (McFie *et al.*, 1950), or in copying tasks they have difficulty coordinating all elements within a common spatial framework (Gainotti and Tiacci, 1970; Warrington *et al.*, 1966). Gardner and his associates suggest that the emotional disturbances after right brain damage may share some similarities (in form at least) to this impaired sense of spatial relationships. In their view right hemisphere patients have a difficulty in integrating emotional perceptions and expressions within the current external situational context, and this leads to inappropriate emotional perceptions and reactions (Winner and Gardner, 1977). A further hypothesised feature of this emotional deficit is that "...right hemisphere patients may have lost the capacity to appreciate the structural relations among emotions, thereby confusing emotions with their opposites..." (Cicone *et al.*, 1980).

To test this latter hypothesis Cicone *et al.* (1980) used a task similar to the emotional face-face matching condition of the present study, where happy and surprised-gleeful faces were categorised as positive emotions; while sad, disgusted, fearful, and angry faces were designated negative. Two error types were distinguished. In an error where emotions of the opposite polarity are confused, a positive emotion is mistaken for a negative emotion; alternatively a negative emotion is incorrectly matched with positive facial expression. Relatively more errors of this type were made by the right lesion group, and Cicone *et al.* concluded "...that there may be in right hemisphere patients a breakdown of the 'spatial' organisation among emotional concepts such that the interrelations among oppositely-toned emotions can no longer be

appreciated." Emotional errors of the same polarity occur when the patient mistakes one positive emotion for another positive emotion; or mistakes one negative emotion with another. These were more frequent after left hemisphere damage, which suggested that the appreciation of the relationships between emotions was relatively spared in the left lesion group.

The present results from the emotional face-face matching condition were not entirely in accord with those of Cicone *et al.* (1980). Right and left hemisphere patients did not differ for errors of the same emotional polarity ( $U=125$ ;  $p=0.26$ ), but in agreement with Cicone *et al.* there was a trend for the right hemisphere patients to make more errors of the opposite emotional polarity ( $U=106$ ;  $p=0.08$ ). This tendency might merely be a secondary consequence of visual perceptual deficit. It is possible that right and left hemisphere patients are equally capable of appreciating emotional "structure", but the right hemisphere patient's facial perceptual disorder introduces a randomising element and makes him particularly prone to erroneously match emotions of opposite polarity. Cicone *et al.* (1980) reject this possibility because they found patients with right hemisphere disease showed the same tendency to make errors of the opposite polarity on a task where emotion was expressed in short written phrases rather than visually. However, as we have seen, scores on the emotional face-word matching condition of the present study are less closely determined by visual perceptual deficit than are emotional face-face matching scores. Cicone *et al.* (1980) would predict emotional errors of opposite polarity to be more common during emotional face-word matching in the right hemisphere group. In contrast, if visual disturbance is responsible for the greater tendency to make errors of the opposite polarity in the emotional face-face matching task, we would predict this tendency would be less evident in the emotional face-word matching

condition. The latter (perceptual) explanation was supported. There was no difference between the right and left hemisphere lesion groups for errors of the same ( $U=154$ ;  $p=0.83$ ), or different emotional polarity ( $U=140$ ;  $p=0.51$ ) during emotional face-word matching.

#### Differential lateralisation of emotion and the facial emotion test

A 3x6 repeated measures analysis of variance was performed, with lesion group (control v right v left hemisphere) as the between subjects factor, while the within subjects factor was emotion type (happy v sad v angry v fear v disgust v surprise), and number of faces correctly identified was the dependent variable. One differential lateralisation of emotion hypothesis predicts a significant interaction between side of lesion and type of emotion. Though this was found [Wilks Lambda=0.48; Approximate  $F(10,78)=3.41$ ;  $p=0.001$ ], figure 13 and further statistical analysis indicate that the interaction was due to the right sided group doing particularly badly with the happy and the fearful faces, and the left sided group having relatively greater difficulty with surprised faces.

The differential lateralisation of emotion hypothesis states that the right hemisphere is specialised for the perception of negative emotion (Sackheim *et al.*, 1982), and as predicted this group had greater difficulty with fear expressions ( $t=2.17$ ;  $p=0.037$ ). However Sackheim *et al.* (1982) would also predict increased right hemisphere impairment for perception of other negative emotions, but there were no significant right-left differences in the perception of sad, angry, or disgusted faces. Furthermore, contrary to expectation the right lesion group had significantly more difficulty than the left lesion group for the perception of happy facial expressions ( $t=3.66$ ;  $p=0.001$ ). Finally, there was a non-significant tendency for patients with left hemisphere lesions to have more difficulty than right hemisphere patients perceiving surprised faces ( $t=1.84$ ;

$p=0.075$ ). The implications of this trend are unclear. Since surprise has received comparatively little attention in the various formulations of the differential lateralisation of emotion hypothesis.

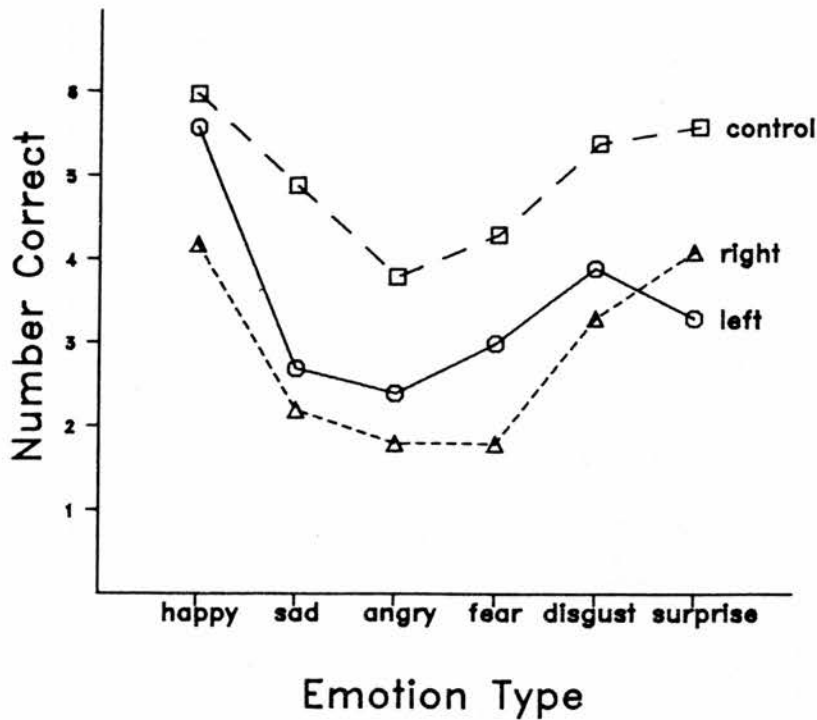


Figure 13

Perception of different types of facially expressed emotion in the right, left and control groups.

There are at least three alternative ways of accounting for the significant interaction found between hemisphere and type of emotion expressed. First, this may be a chance occurrence, a statistical artifact. A second possibility to be considered is that emotional functions are differentially lateralised but the Sackheim *et al.* (1982) version is incorrect. However the present data from the test of perception of facial emotion do not appear to suggest an immediately

obvious and parsimonious alternative form of differential lateralisation of emotion hypothesis. Thirdly, these effects may have something to do with the visual properties common to happy, fearful, and surprised facial expressions. For some reason right hemisphere visual-perceptual functions may be especially well suited to perceiving happy and fearful expressions, and the left hemisphere may be particularly good at perceiving surprised facial expressions in the perception of facial emotional task.

### Conclusions

1. Patients with right and left hemisphere lesions were both impaired with respect to controls on the test of perception of facially expressed emotion. Facial emotional perception was least affected in the left posterior group.
2. In the right hemisphere group face-face matching scores were most impaired in patients with visual/perceptual deficit, while in the left hemisphere group face-word matching scores were particularly associated with presence of verbal-cognitive deficit. It is concluded that impaired facial emotional perception is, at least partially, due to different perceptual/cognitive deficits in the right and left hemisphere patients.
3. Two experimental strategies aimed to dissociate the contributions of specifically emotional and of perceptual/cognitive deficit to facial emotional perception scores. Though neither strategem was successful, the data were in fair agreement with the attentional/emotional hypothesis. While the focal/diffuse dichotomy fitted the data reasonably well, the views of Gardner and his associates were not supported.
4. The hypothesis that the right hemisphere is dominant for perception of negative emotion and the left hemisphere is specialised for perception of positive emotion was disconfirmed.

## 6.2. Test of Perception of Emotion Expressed in Speech and Text

Table 24 gives the distribution of total scores for the test of perception of emotion expressed in speech and text by the 10 control, 23 right hemisphere and 17 left hemisphere patients. In a one-way analysis of variance with total scores as the dependent variable the group effect was significant [ $F(2,38)=10.71$ ;  $p=0.0002$ ]. Post hoc Scheffe tests showed right and left hemisphere groups each made significantly more errors than controls. However total scores of the hemispheric groups were not significantly different.

<u>Score Range</u>	<u>Right Hemisphere</u>	<u>Left Hemisphere</u>	<u>Controls</u>
65-60	1	1	5
59-54	1	2	1
53-48	3	4	4
47-42	4	4	
41-36	4	2	
35-30	1	1	
29-24	4	3	
23-18	1		

Table 24

Total scores on the test of emotion expressed in speech and text for the control, right and left hemisphere groups.

The hypothesis that emotional functions are differently lateralised in the male and female brain predicts a significant interaction between sex and side of lesion. Therefore sex and hemisphere (right v left) were made factors in a 2x2 analysis of variance, where total scores on perception of emotion expressed in speech and text was the dependent variable. However the sex by

hemisphere interaction was not significant [ $F(1,32)=0.35$ ;  $p=0.56$ ], and neither sex nor side of lesion approached significance. For subsequent analyses the verbal emotion scores of the 24 male and the 22 female subjects are treated as equivalent.

### Perception of emotion expressed in speech and text with lesion size, type and location

**Lesion size and aetiology:** The hemispheric groups were fairly well matched for lesion size and type, but to make the groups a little more comparable, patients with arteriovenous malformations or a metastasis were excluded (3 right and 3 left hemisphere) and the effect of lesion size was partialled out in a one-way analysis of covariance. The effect of the lesion size covariate did not reach significance [ $F(1,32)=2.06$ ;  $p=0.16$ ], and lesion side did not significantly determine total perception of verbal emotion scores [ $F(1,32)=0.37$ ;  $p=0.55$ ].

**Lesion location:** Figure 14 gives mean scores for the right anterior ( $N=3$ ), right posterior ( $N=9$ ), left anterior ( $N=4$ ), left posterior ( $N=7$ ), and control ( $N=10$ ) subgroups. A one-way analysis of variance was performed to make a preliminary assessment of the relative performance of the 4 lesion subgroups and controls. The group effect was highly significant [ $F(4,28)=12.41$ ;  $p<0.0001$ ]. Post hoc paired comparisons using the Newman-Keuls procedure showed that the left anterior, right anterior, and right posterior patients' scores were significantly lower than those of the control or left posterior subgroup. There was no significant difference between the left posterior group and controls, though there was a trend to greater deficit after left posterior damage ( $U=14.5$ ;  $p=0.09$ ).

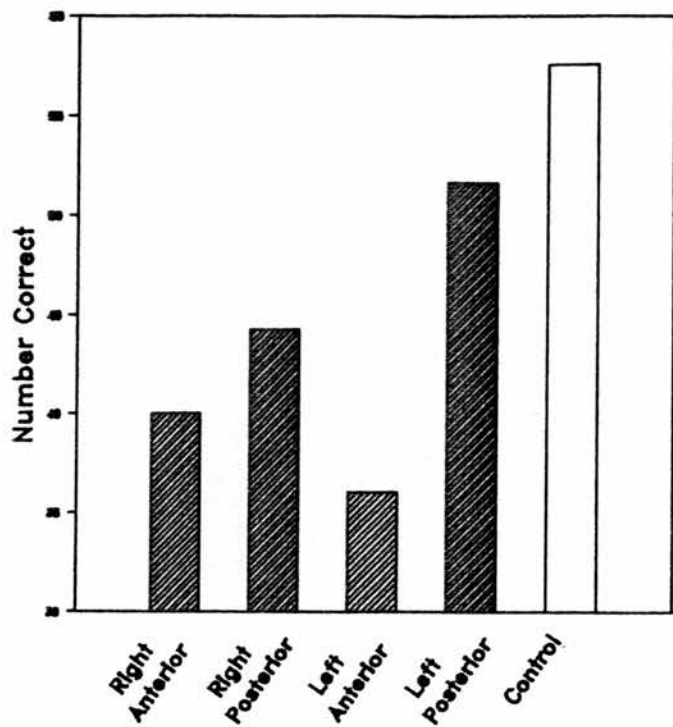


Figure 14

Total scores for perception of emotion expressed in speech and text for right anterior, right posterior, left anterior, left posterior, and control subgroups.

On the statistical model where main effects are additive, the intra- and inter-hemispheric effects summate arithmetically to produce the pattern of differential impairment in verbal emotion perception between the 4 lesion subgroups seen in figure 14. To test the explanatory power of this additive model a 2x2 analysis of variance was performed, with the intra- and inter-hemispheric dimensions as factors, and total verbal emotion scores as the dependent variable. The intra-hemispheric factor was significant; patients with anterior lesions made significantly more errors than those with posterior lesions [ $F(1,19)=12.34$ ;  $p=0.002$ ]. The effect of side of lesion did not achieve significance [ $F(1,19)=2.11$ ;  $p=0.16$ ]. However the additive model may not provide



the best fit to the data, as there was a non-significant interaction between the intra- and inter-hemispheric factors [ $F(1,19)=3.77$ ;  $p=0.067$ ]. Figure 14 suggests that the non-significant interaction between the intra-hemispheric factor and the side of lesion factor was due to the highest and the lowest scoring subgroups being both within the left hemisphere.

(a)	<u>Right</u> <u>Hemisphere</u>	<u>Left</u> <u>Hemisphere</u>	<u>U</u>	<u>p</u>
Tone-Face	4.4	6.5	72.0	0.004
Content-Face	7.8	8.4	141.0	0.53
Content+				
Tone-Face	5.2	6.8	115.0	0.09
Tone-Word	5.9	6.8	128.5	0.30
Content-Word	9.2	8.4	136.5	0.43
Content+				
Tone-Word	7.5	6.5	126.5	0.27

(b)	<u>Right</u> <u>Hemisphere</u>	<u>Left</u> <u>Hemisphere</u>	<u>t-value</u>	<u>p</u>
<b>Total score</b>	40.1	43.6	0.97	0.34
<b>Response</b>				
Face Match	17.5	21.9	2.41	0.022
Word Match	22.6	21.7	0.38	0.70
<b>Stimulus</b>				
Tone	10.3	13.3	2.51	0.017
Content	17.0	16.8	0.12	0.91
Content+Tone	12.7	13.5	0.50	0.62

Table 25

(a) Mean scores for the 6 subtests of the test of perception of verbally expressed emotion. The Mann-Whitney U test was used to compare right and left hemisphere group scores. (b) The 6 subtests are summated to give total score. The 3 face matching subtests are summed in the verbal-face response condition, and the word matching subtests are added together for the verbal response condition. Similarly, for the tone stimulus condition, the tone-face and tone-word subtests are summed. The t-test was used to compare scores of the right and left hemisphere groups.

It is interesting that these results are in agreement with the main one-way analyses of variance using larger groups, where perception of verbally expressed emotion was found to be significantly depressed after anterior lesions but no right-left were demonstrated. However in these analyses it was not possible to evaluate the significance of the interaction between the inter- and intra-hemispheric factors, as these factors were not considered together. Of course, subgroup sizes are low, and the present evidence for an interaction is unreliable. Furthermore, a parsimonious explanation for this interaction does not immediately present itself. Nevertheless, Robinson and Szelata (1981) have suggested a special association between emotional disorder and left anterior lesions.

#### Cognitive/perceptual asymmetries and perception of emotion expressed in speech and text

Three categories of stimulus were presented (emotion expressed in tone of voice, semantic content, or tone + content), and two types of response requested (face or word matching). All of the possible combinations of stimulus and response conditions were assessed, and table 25a compares performance of the right and left hemisphere groups on each of these 6 component tasks. The right lesion group was significantly more impaired when emotion expressed in tone of voice was matched with emotional faces. This group also tended to show greater impairment matching faces with utterances where emotion was expressed in both tone and content. There were no significant right-left differences on the 4 other subtests.

**Right hemisphere perceptual-cognitive deficit:** Total face matching score was the sum of the 3 face matching subtests, while the 3 word matching subtest

scores were combined to give total word matching score. The right hemisphere group were impaired relative to the left lesion group for total face matching score (Table 25b). Some of these right hemisphere patients also showed evidence of visual perceptual deficit, including neglect and impairment of facial recognition. Therefore patients with visual disturbance (N=8) were compared with right hemisphere patients who gave no indication of clinically significant visual perceptual disturbance (N=11). Verbal-face matching was more impaired in the patients with visual disturbance ( $U=18$ ;  $p=0.033$ ). In contrast, patients with and without visual perceptual deficit did not differ significantly for the verbal-word matching condition ( $U=27.5$ ;  $p=0.18$ ), presumably because the latter emotional verbal-word matching tasks are less reliant on intact facial perception. Table 26 also suggests a relatively weak association between degree of visual perceptual deficit and emotional verbal-face matching. In the right hemisphere group visual perceptual/cognitive score (face identity recognition, degree of neglect, and perhaps Performance IQ) tend to have higher correlation coefficients with the verbal-face matching scores than with verbal-word matching performance.

Of course, the relationship between visual perceptual/cognitive scores and verbal-face matching is less clear cut than the link between visual perceptual/cognitive scores and the emotional face-face matching condition of the test of perception of facially expressed emotion (contrast tables 26 and 23). However emotional face-face matching is likely to be more heavily dependent on capacity for facial perception, than emotional verbal-face matching. Presumably, emotional verbal-face matching requires left hemisphere language processes in addition to the capacity for face recognition. Therefore emotional verbal-face matching is likely to be impaired after both right and left hemisphere damage. Consequently, if any right-left difference were found for

the emotional verbal-face matching condition, we would expect it to be less pronounced than the right-left differences on face-face matching tasks.

	RIGHT HEMISPHERE			LEFT HEMISPHERE	
	Verbal-Face	Verbal-Word		Verbal-Face	Verbal-Word
Performance IQ	0.71**	0.64**		0.34	0.44
Verbal IQ	0.67**	0.38		0.42	0.69**
Face Identity Recognition	0.83**	0.60		-	-
Recognition Memory					
Faces	0.51*	0.67**		0.13	-0.02
Words	0.57*	0.43		0.47	0.17
Visual Neglect					
Line Cancellation	-0.50*	-0.46		-	-
Line Bisection	-0.40	-0.16		-	-
Token Test	-	-		0.53	0.85***

Table 26

Correlations between general sensori-motor-cognitive measures and response manipulations for the test of perception of verbally expressed emotion. There is a tendency for verbal-word matching to have a close link with measures of verbal-cognitive function in left hemisphere group. The association between verbal-face matching and visual-perceptual-cognitive measures is not marked in the right hemisphere group. (\*\*\* -  $p < 0.001$ ; \*\* -  $p < 0.01$ ; \* -  $p < 0.05$ )

The 6 subtests comprising the test of perception of verbally expressed emotion were combined differently to produce scores for stimulus mode. For example, the score for ability to perceive emotion expressed in tone of voice was the sum of the tone-face matching plus the tone-word matching measure. Three such stimulus mode measures were constructed, assessing ability to

perceive emotion expressed in (a) tone of voice, or (b) in content, or (c) in tone and content.

Table 25b shows that patients with right hemisphere disease have particular difficulty perceiving emotion conveyed by tone of voice. This problem in perception of emotional tone appeared to be dissociated from visual perceptual symptoms. Patients with or without visual disturbance did not differ in their ability to perceive emotional tone ( $U=25.5$ ;  $p=0.13$ ). Also, table 27 shows that in the right hemisphere group perception of emotional tone was significantly associated with Performance IQ, but not with either face identity recognition or the measures of visual neglect.

The dissociation between visual perceptual disturbance and emotional tone perception deficits is, perhaps, a consequence of the relative anatomical separation of the auditory and visual systems (Brodal, 1981). Also, modality specific perceptual/cognitive deficits have been suggested from human (Warrington and Shallice, 1984; Beauvois *et al.*, 1978), and animal work (Mishkin, 1979; Gross, 1973). It is therefore hypothesised that in order to establish an association between general sensory/perceptual deficit and impaired perception of emotional tone of voice, measures of capacity to make complex perceptual judgements in the visual modality are inadequate. However the deficit in identification of pitch associated with right hemisphere disease (Sidtis, 1984), may have a considerably closer connection with impairment in the perception of emotion expressed through tone of voice.

Visual perceptual deficit also appeared dissociable from ability to perceive emotion conveyed in verbal-semantic content. Patients with and without visual disturbance did not differ in their ability to perceive emotion expressed in written sentences ( $U=28$ ;  $p=0.21$ ). Table 27 shows perception of emotion

expressed in verbal content did not appear to have a selective or specific relationship with measures of perceptual-cognitive deficit in the right hemisphere group. Thus Verbal IQ correlated with emotional content score, while neither of the measures of visual neglect were significantly associated with ability to perceive emotion expressed in verbal content (Table 27).

	RIGHT HEMISPHERE		LEFT HEMISPHERE	
	Tone	Content	Tone	Content
Performance IQ	0.62**	0.61**	0.57*	0.32
Verbal IQ	0.30	0.67**	0.79**	0.51
Face Identity Recognition	0.59	0.71*	-	-
Recognition Memory				
Faces	0.54*	0.49	0.14	0.28
Words	0.47	0.47	-0.11	0.22
Visual Neglect				
Line Cancellation	-0.34	-0.42	-	-
Line Bisection	-0.21	-0.28	-	-
Token Test	-	-	0.76**	0.60*

Table 27

Correlations between general sensori-motor-cognitive measures and stimulus manipulations in the test of perception of verbally expressed emotion. In the left hemisphere group there is a tendency for tone and content scores to be related to Verbal IQ and Token Test scores. No obvious link between perceptual-cognitive measures and tone, or content scores was noted in the right hemisphere group. (\*\*- $p < 0.01$ ; \*- $p < 0.05$ )

**Left hemisphere verbal-cognitive deficit:** In spite of the left hemispheric specialisation for language processes, mean emotional verbal-word matching

scores were not significantly different in the left and right hemisphere groups (Table 25b). Nevertheless, the left hemisphere group performance was deficient, when compared with the control group ( $t=4.29$ ;  $p<0.001$ ). This deficit in emotional verbal-word matching in the left hemisphere group was related to presence of dysphasia ( $U=3.5$ ;  $p<0.0003$ ). Dysphasic ( $N=10$ ) patients also tended to be impaired relative to the non-dysphasic subgroup ( $N=8$ ) for emotional verbal-face matching scores ( $U=18.5$ ;  $p=0.055$ ). Table 26 provides further evidence that verbal-cognitive deficit contributes to impairment on the test of verbally expressed emotion. Token Test scores and Verbal IQ correlate significantly with emotional verbal-word matching scores, but not with emotional verbal-face matching scores.

Despite the relative superiority of the left hemisphere group for perception of emotion expressed in tone of voice (Table 25b), when compared with the controls, perception of emotional tone was impaired in the left hemisphere group ( $t=2.84$ ;  $p=0.009$ ). Impairment in the perception of emotional tone after left lesions was closely related to the presence of dysphasia ( $U=2.5$ ;  $p=0.0002$ ). This is confirmed in table 27, which shows that Verbal IQ and Token Test score were also significantly related to ability to perceive emotional tone. In other studies deficits in perception of emotional tone of voice (Seron *et al.*, 1982; Schlanger *et al.*, 1976), and meaningful sounds (Varney, 1980; Spinnler and Vignolo, 1966) have been associated with dysphasia.

Similarly, though the left hemisphere group were not impaired relative to the right hemisphere group for perception of emotion expressed in verbal-semantic content (Table 25b), the left hemisphere group was deficient on this measure when compared with controls ( $t=3.61$ ;  $p=0.001$ ). Errors in the left hemisphere patients were related to the presence of dysphasia ( $U=13.5$ ;  $p=0.016$ ), and emotional content score was significantly related to performance

on the Token Test (Table 27), and non-significantly associated with Verbal IQ ( $r=0.51$ ;  $p=0.08$ ).

It has been suggested that deficits on the test of perception of verbally expressed emotion may be determined by auditory (and perhaps weakly by visual) perceptual impairment in the right hemisphere group, and verbal-cognitive losses after left hemisphere damage. There are two problems with this hypothesis. First, facial and word recognition memory scores appear to consistently deviate from the expected pattern. For example, in table 26 we might have expected word recognition memory to correlate significantly with emotional verbal-word matching in the left hemisphere group, while face recognition memory would be most closely related to emotional verbal-face matching in the right hemisphere group. However it is not particularly uncommon to find individual patients with memory disorders in the relative absence of cognitive or perceptual deficit (e.g. Winocur *et al.*, 1984; Milner *et al.*, 1968). Indeed, it is generally accepted that, while some memory and cognitive processes may be related (Butters and Cermak, 1975), certain memory and perceptual/cognitive functions are dissociable (e.g. Meudell and Mayes, 1982). It is therefore not surprising that the degree of association between verbal emotional perception impairment and perceptual/cognitive losses, differs from the level of association between memory deficit and verbal emotional perception deficits.

The hypothesis, that different perceptuo-motor and cognitive deficits in each hemisphere influence performance on the perception of verbal emotion test, confronts a second problem. The prediction that the left hemisphere group would have greater difficulty than right lesion patients on the emotional verbal-word matching condition was not confirmed (Table 25b). Nevertheless Kolb and Taylor (1981) did find the expected selective left hemisphere deficit



for a similar task where the patient had to name the emotion expressed in written material. It is possible that their task made greater demands on verbal skills and so was more sensitive to dysphasic deficit. In the present study the investigator repeatedly corrected a dysphasic patient if they misread the emotional sentence. Had these reading errors been left uncorrected, a selective deficit in the verbal-word matching condition after left hemisphere damage might have been found. Indeed, Cicone *et al.* (1980) coached patients in this way in a test where emotion was expressed in short written phrases, and no significant difference between right and left hemisphere groups was found. Furthermore, in the present study, to discourage subjects from using verbal mediators during the emotional face matching conditions, the face matching condition was administered first. By the time the emotional verbal-word matching tests were administered the patient was well practiced. Such practice might tend to raise the left hemisphere dysphasic patients' performance, making it more comparable to the relatively spared performance of the right hemisphere patients. In this way practice effects would tend to obscure a selective left hemisphere deficit on the emotional verbal-word matching tasks.

Right hemisphere dominance for emotion and the test of perception of emotion expressed in speech and text

Emotional verbal-word matching scores of the left hemisphere group were impaired relative to controls. Though "coaching" and practice may possibly have obscured a right-left difference in emotional verbal-word matching scores, practice did not eliminate the association between dysphasia and emotional verbal-word matching scores. Clearly, if language impairment causes some deficit in the left hemisphere group, then the absence of a right-left difference in emotional verbal-word matching scores implies that the right

hemisphere group performance is also deficient. In confirmation, the right hemisphere group were impaired relative to controls for emotional verbal-word matching. Conceivably it is a deficit in **emotional** perception that affects performance in the right hemisphere group, while verbal-cognitive deficit is largely responsible for impairment after left hemisphere damage. Indeed, Wechsler (1973) previously suggested this possibility when he found that right hemisphere patients showed particular difficulty in verbal recall of an emotional short story. Of course, the existence of a right hemisphere emotional specialisation is not established, and there is no general agreement about the neurophysiological or neuropsychological bases of the hypothesised right hemisphere emotional function. However we should consider the extent to which the present data can be explained by current views about the nature of right hemisphere emotional dominance.

**Right hemisphere attentional/emotional specialisation:** Scores on the line cancellation and bisection tasks generally tended to correlate weakly and non-significantly with several of the measures contained within the test of perception of verbally expressed emotion (Tables 26 & 27). However it has been argued that visual perceptual deficits other than visual neglect sometimes impair line bisection and cancellation performance, therefore in a subsequent analysis the 6 patients with clear neglect were compared with the 13 right hemisphere patients without neglect (the 2 patients with marked visual perceptual disturbances were included in the non-neglecting group). The neglecting group were significantly impaired for emotional verbal-word matching ( $U=12.5$ ;  $p=0.017$ ), for perception of emotion expressed in verbal content ( $U=15$ ;  $p=0.037$ ), and for verbal-face matching ( $U=15$ ;  $p=0.037$ ). There was also a tendency for the neglecting group to perform less well at emotional tone perception ( $U=18.5$ ;  $p=0.072$ ).

It is intriguing that patients with visual neglect should show greater impairment on most measures of perception of verbal emotion, whereas only one of these comparisons between patients with and without visual disturbance approached significance. This might suggest that deficits in the test of verbal emotional perception are weakly related to visual problems *per se*, but are strongly associated with the attentional/emotional deficit which underlies the symptoms of neglect (Heilman *et al.*, 1978). However this conclusion must be viewed with considerable caution. Lesion size tended to be larger ( $U=17.5$ ;  $p=0.058$ ) in the neglecting group (122.3cc) compared with the non-neglecting group (73.3cc). Also, patients with neglect were impaired relative to the non-neglecting group in Performance IQ ( $U=0$ ;  $p<0.0001$ ). (Though not for Verbal IQ, Benton Visual Retention Test scores, or face and word recognition memory.) Also, only 2 patients were removed from the group with visual perceptual deficit to provide a group of patients with neglect. Similarly, the group without neglect was identical to the group without visual disturbance, except that 2 patients with visual disturbance were added. When group sizes are low small changes in their constitution can produce misleading variations in significance levels.

**Differential hemispheric functional organisation:** In the right hemisphere the anterior and posterior subgroups did not differ significantly for total score on the test of perception of verbally expressed emotion, but scores were significantly different in anterior and posterior subgroups in the left hemisphere (See figure 14). This is consistent with the hypothesis that functions are diffusely represented in the right hemisphere, and more focally organised in the left hemisphere. However right hemisphere test intercorrelations should also be higher than the test intercorrelations within the left hemisphere group. Though this effect is not marked in table 26, correlation coefficients between

memory tests and the response conditions do tend to be higher in the right hemisphere group. Also, 8 of these right hemisphere correlations compared with only 2 of the left hemisphere correlation coefficients achieved significance. On the other hand, table 27 is not consistent with the focal/diffuse dichotomy. There was not a general tendency for correlation coefficients to be higher, and only 5 out of 14 correlation coefficients were significant in the right hemisphere group, whereas in the left hemisphere group 5 out of 10 coefficients were significant.

**Emotional integrations of the right hemisphere:** Gardner and his associates have made special study of emotion expressed linguistically. Brownell *et al.* (1984) hypothesise that the emotional significance of verbal material is conveyed through connotative and denotative meanings. The denotation of a word is given by its dictionary definition. For example, the word "book" denotes an object made of paper and cardboard, with numbered pages, and chapters. In left hemisphere patients, the capacity to understand emotion in speech and text is impaired due to dysphasia, and to selectively impaired comprehension of denotation. The connotations of words are more oblique and elusive, in addition these meanings may be emotionally charged. Thus the word "book" has connotations of studying and learning, there are associated images (and connotations) of pleasurable fictional reading, and negative emotional associations with difficult reading. Brownell *et al.* (1984), Wapner *et al.* (1981), and Winner and Gardner (1977) believe that patients with right hemisphere lesions are insensitive to connotative meanings.

This theory can account for the present finding that right and left hemisphere damage both significantly impair performance on emotional verbal-word matching. However these workers have also argued that right hemisphere patients are liable to confuse positive with negative emotions.

Cicone *et al.* (1980) claim this disorganisation in the structure of emotions occurs "across the board". We have seen that in both visual and linguistic tasks they found right hemisphere patients tended to make more errors of opposite polarity while left lesion patients' errors tended to be between emotions of the same polarity. The present data from the test of perception of verbally expressed emotion did not confirm this hypothesis. In a repeated measures analysis of variance with lesion side (right v left hemisphere), and error type (opposite v same polarity) as factors, the predicted interaction between lesion side and error type was not found [ $F(1,34)=1.13$ ;  $p=0.30$ ].

#### Differential lateralisation of emotion and the test of perception of emotion expressed in speech and text

In the test of verbal emotional perception 23 items expressed anger, 20 expressed happiness, and 18 expressed sadness. Patient's raw score for each emotion type was converted to a percentage correct score. Percentage correct scores were made the dependent variables in a 3x3 repeated measures analysis of variance, where lesion group (control v right v left hemisphere) was the between subjects factor, and emotion type (happy v angry v sad) the within subjects factor. The differential lateralisation of emotion hypothesis predicts a significant interaction between lesion group and emotion type; where negative emotional perception is most impaired after right hemisphere damage and positive emotion is selectively deficient after left hemisphere damage. However the interaction between lesion side and emotional type did not achieve significance [Wilks Lambda=0.8839; Approximate  $F(4,84)=1.34$ ;  $p=0.26$ ]. In fact, figure 15 shows that the differential lateralisation theory was directly contradicted, as right hemisphere damage produced a significantly greater deficit in the perception of happy stimuli ( $t=2.21$ ;  $p=0.03$ ).

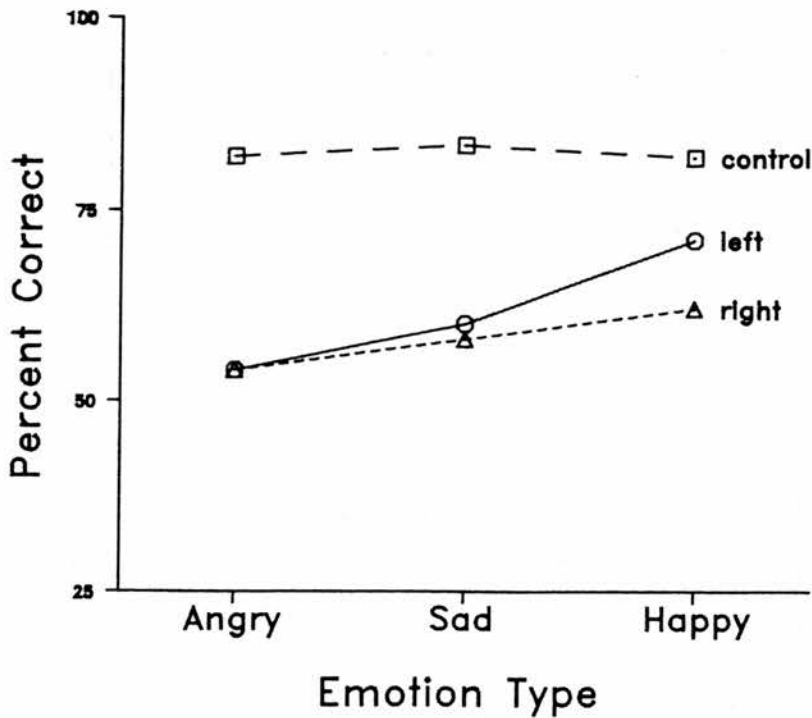


Figure 15

Comparison of perception of 3 types of emotion expressed in speech and text by right hemisphere, left hemisphere, and control groups.

### Conclusions

1. While perception of emotion expressed in speech and text appeared to be least impaired in the left posterior group, it is likely that performance is impaired in all 4 lesion subgroups.
2. Impaired performance on the test of verbally expressed emotion appeared partially determined by dysphasia in the left hemisphere group. There was a non-significant association between facial perceptual deficits in the right lesion group and patients' performance on one component of the test of perception of verbally expressed emotion, but auditory perceptual deficits may make a greater contribution to impaired emotional perception in this mode.
3. These data did not offer strong support for the hypothesis of right hemisphere dominance for the perception of emotion. However findings were fairly consistent with the attentional/emotional hypothesis of Heilman and his collaborators. Support for the focal/diffuse dichotomy was equivocal.

4. In some respects the present findings are in agreement with those of Gardner and his associates, who hypothesise that the right hemisphere is specialised for the appreciation of connotative meanings. However in other important respects these data are in conflict.
5. The differential lateralisation of emotion hypothesis as stated by Sackheim *et al.* (1982) was disconfirmed.

### 6.3. Discussion

Investigations of the neural substrate of emotion have tended to consider either the intra-hemispheric factor or the inter-hemispheric factor in isolation. Indeed, this approach was followed in the main analyses of the present investigation. This separate assessment of the contributions of the intra- and inter-hemispheric factors implicitly assumes an additive statistical model, where inter-hemispheric and intra-hemispheric main effects summate arithmetically. This assumption may not be valid, as it is possible that emotional deficit is greatly enhanced after damage to a particular neural site.

In point of fact, the distribution of total perception of facial emotion scores was best explained by the additive model. This was, however, not the case for total perception of verbally expressed emotion scores, as there was a near significant interaction between inter- and intra-hemispheric factors, the groups with the highest and lowest total scores both lying within the same (left) hemisphere. Of course, a near significant interaction only raises a doubt about the validity of the approach where inter- and intra-hemispheric dimensions are considered separately. In any case, there were too few patients in the right anterior and left anterior subgroups to allow an adequate test of the significance of the interaction term. Furthermore, the validity of the comparison between the two statistical models is uncertain because lesion aetiology was



not homogeneous for the 4 lesion subgroups. The left posterior subgroup included 2 patients with removal of sphenoid wing meningiomas, 3 patients with AVM excision, one stroke patient and only one patient with a glioma. In the other 3 subgroups lesions were generally caused by intrinsic tumours. Nevertheless, in this study the results have generally remained essentially unaltered when it has been possible to introduce partial controls for lesion variables.

For the test of perception of facially expressed emotion all 4 lesion subgroups were impaired relative to the controls. Total perception of verbal emotion scores were significantly depressed in 3 of the lesion subgroups, and there was a non-significant tendency for left posterior patients to show impairment when contrasted with controls. Though perception of emotion scores were depressed by damage in several neural regions, it is likely that the cause of deficit varied with lesion site. Thus impairment of emotional perception after frontal lesions may stem from disturbance of emotional functions represented in anterior regions. Furthermore, there were indications that total emotional perception scores were depressed by different factors in the right and left hemispheric groups. It was concluded that the deficit in the perception of emotion tests of left hemisphere patients is at least partially mediated by a language-cognitive disturbance. Similarly the characteristic right hemisphere perceptual-cognitive disturbances appeared to selectively impair performance on the emotion subtests that were most dependent on visual perception.

Right and left hemispheric perceptual/cognitive deficit might contribute to impaired emotional perception in two ways. First, deficits in emotional perception may be secondary manifestations of primary perceptual/cognitive disturbances. For example, the oculo-motor deficits associated with right



posterior damage (Chedru *et al*, 1973) may impair a patient's capacity to scan the emotional face and consequently lead to his misinterpreting a facial emotional expression. Similarly, word finding difficulties or other dysphasic deficits may be responsible for impairment in the left hemisphere group. Secondly, there may be a more intimate or basic link between the perceptual/cognitive deficits and emotional perception deficits of the right and left hemisphere groups. On this view, the right and left hemispheres are specialised for the perception of different aspects of the incoming sensory stimulus. The left hemisphere performs a "conceptual" analysis, and the right hemisphere analyses complex "perceptual" features. These right-left differences in perceptual processes may be manifest in all sensory modalities (Vignolo, 1982; Milner and Taylor, 1972; Faglioni *et al*, 1969), and it has also been suggested that different aspects of facial stimuli are analysed by each hemisphere (Damasio *et al*, 1982; Levy *et al*, 1972). Warrington and her colleagues have considered this possibility in some detail for the process of object recognition (Warrington and Shallice 1984; Warrington and Taylor, 1978; Warrington, 1975; Warrington and Taylor, 1973; Taylor and Warrington, 1973).

Warrington and Taylor (1978) hypothesise that the right hemisphere is specialised for perceptual categorisation, which is a process concerned with recognising the invariant visual features of an object. For example, the visual stimulus falling on the retina transforms dramatically as an external object rotates, yet we can group together these different images as being the several manifestations of a single object. Extending this model to perception of facial emotional expressions, it might be hypothesised that the process of perceptual categorisation is essential in the recognition of facial emotional expressions. For example, smiles or angry expressions vary with the situational context as well as between individuals. In spite of these differences we are able to identify

a fundamental unity in the facial configuration of each type of expression, and the right hemisphere may be specialised for the perceptual categorisation of facial emotional expression.

Warrington and Taylor also (1978) hypothesise that objects are semantically categorised in the left hemisphere. Semantic categorisation is the process where the percept of the object is associated with semantic meanings. For example, the percept of a chair would be allocated to the semantic field of inanimate objects, it would also be associated with specific functions e.g. it is for sitting on, it may have a decorative function. Emotional facial expressions also acquire functional meanings. For example, angry expressions and disgusted expressions might both be placed in a semantic category of "negative behaviours". Alternatively the percept of a wink, and of a smile might be allocated to the semantic categories of "friendly", and also "sociable", behaviours. It may be that the left hemisphere is specialised for this process of semantic categorisation of emotional facial expression.

### 7.1. Spontaneous Facial Movement in a Failure Situation

The patient's reactions were video-recorded while a modified version of the Wisconsin Card Sorting Test was administered. The number of FACS action units produced in reaction to failure was counted, according to a previously described procedure. In a 3x2 analysis of variance, total number of FACS action units produced in 10 reactions to failure was the dependent variable; with lesion group (right v left v control), and sex as factors. The lesion group effect fell short of significance [ $F(2,33)=3.14$ ;  $p=0.056$ ]. The sex main effect [ $F(2,33)=0.10$ ;  $p=0.92$ ], and the sex by hemisphere interaction [ $F(2,33)=0.52$ ;  $p=0.60$ ] were not significant. Qualitative, self-report, and heart-rate measures were also made during the card sorting task, but in no case did the effect of sex, or of the sex by hemisphere interaction achieve significance, and so the 20 males and the 19 females were grouped together.

FACS action units produced by 8 control, 16 right hemisphere, and 15 left hemisphere patients are given in table 28. In a one-way analysis of variance the group effect was significant [ $F(2,36)=3.34$ ;  $p=0.047$ ]. Post hoc testing with the Newman-Keuls procedure showed facial movement was reduced in the right hemisphere group relative to controls. No other paired comparison achieved significance, but table 28 and further statistical analysis reveals a clear tendency for the left lesion group to produce fewer FACS action units than controls ( $t=2.33$ ;  $p=0.03$ ).

<u>Action Units</u>	<u>Right Hemisphere</u>	<u>Left Hemisphere</u>	<u>Controls</u>
36-33			1
32-29	1		
28-25		3	3
24-21	1	1	1
20-17	4	1	1
16-13	4	4	2
12-9	2	2	
8-5	2	2	
4-0	2	2	

Table 28

Total number of FACS action units produced during 10 reactions to failure, in the hemispheric and control groups.

The upper face appears to be bilaterally represented at the cortex, whereas direct cortical control of the lower face is unilateral (Rinn, 1984). However right and left lesion groups did not differ in frequency of upper or lower face movements (Table 29). Table 29 also shows that comments and vocalisations occurred with equal frequency in the right and left lesion groups. Though significantly fewer head movements were made in the right lesion group, no interjudge or test-retest reliability data is available for this latter measure.

#### Spontaneous facial movement with lesion size, type and location

**Lesion size and aetiology:** Right hemisphere lesions were slightly larger, and the hemispheric groups were not precisely matched for lesion aetiology. Therefore all metastases and arteriovenous malformations were excluded (3 right and 3 left hemisphere), and total lesion size was partialled out in a one-way analysis of covariance, but the hemispheric groups still did not differ in frequency of spontaneous facial movement [ $F(1,22)=0.12$ ;  $p=0.74$ ].

	<u>Right</u> <u>Hemisphere</u>	<u>Left</u> <u>Hemisphere</u>	<u>t-value</u>	<u>p</u>
Upper Face	4.9	6.5	0.92	0.36
Lower Face	9.9	7.9	0.92	0.37
Head Movements	3.6	6.2	2.12	0.043
Comments	1.9	3.1	-	0.77
Vocalisations	1.4	1.9	-	0.18

Table 29

Mean number of component movements produced by right and left hemisphere lesion groups after being told they had made an incorrect card sort. Mann-Whitney U test was used where t-values are absent.

**Lesion location:** The mean scores for the 4 lesion subgroups and control patients are represented in figure 16. In a one-way analysis of variance the group effect was significant [ $F(4,24)=4.54$ ;  $p=0.007$ ]. Post hoc testing with the Newman-Keuls procedure showed that the right anterior ( $N=3$ ) and left anterior ( $N=3$ ) groups each produced significantly fewer FACS action units than right posterior ( $N=8$ ), or left posterior ( $N=7$ ), or control ( $N=8$ ) groups. No other paired comparisons between subgroups was significant, therefore there was a selective reduction of facial movement in both anterior lesion groups during the card sorting test situation.

This distribution of mean facial movement scores in the 4 lesion subgroups is best fitted by an additive statistical model. In a 2x2 analysis of variance patients with anterior lesions made significantly fewer FACS action units [ $F(1,17)=15.10$ ;  $p<0.001$ ], but there were no right-left differences in facial

movement [ $F(1,17)=0.16$ ;  $p=0.70$ ], and the interaction between inter- and intra-hemispheric factors was not significant [ $F(1,17)=0.57$ ;  $p=0.46$ ].

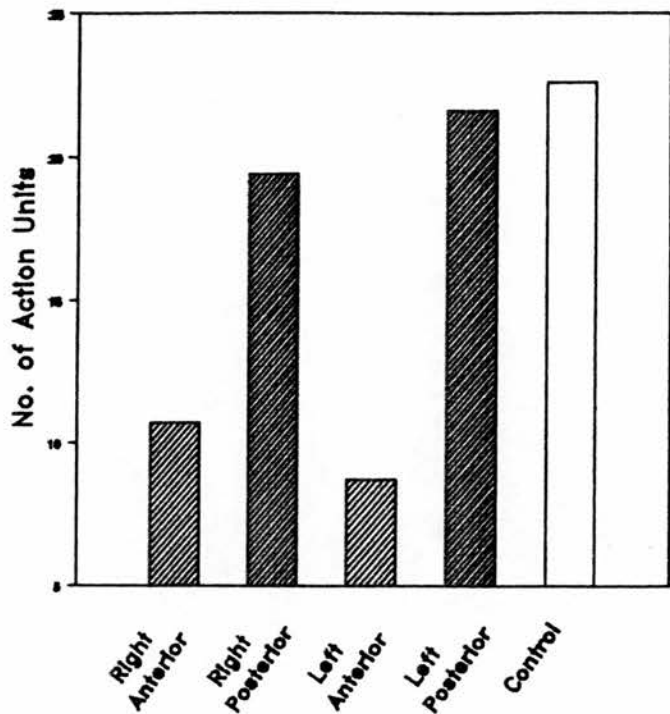


Figure 16

Number of FACS action units produced by right anterior, left anterior, right posterior, left posterior, and control patients.

#### Relationship between general sensori-motor deficit and facial movement after failure

**Left hemisphere cognitive-motor deficit:** Kimura (1982 & 1979) discusses the strong evidence for the lateralisation of specialised motor control functions in the left hemisphere. Left hemisphere damage is associated with ideational and

ideomotor apraxia (De Renzi *et al.*, 1980; De Renzi *et al.*, 1968), deficits in copying meaningless manual movement sequences (Jason, 1983a&b; Kimura and Archibald, 1974), and perhaps "executive" constructional apraxia (Warrington *et al.*, 1966). Facial movement disorders such as oral apraxia (De Renzi *et al.*, 1966) and inability to imitate meaningless oral-facial movement sequences also occur after left hemisphere disease (Mateer and Kimura, 1977). These facial and manual movement deficits are generally associated with dysphasia (Kimura, 1982), and in the present study the 9 patients with dysphasia made more errors in imitation of facial movement sequences when compared with the 7 left hemisphere non-dysphasic patients ( $U=3$ ;  $p=0.002$ ), or the right hemisphere group ( $U=9$ ;  $p=0.001$ ). Furthermore, imitation of facial movement was significantly related to Token Test score ( $r=-0.81$ ;  $df=11$ ;  $p=0.001$ ) and Verbal IQ ( $r=-0.68$ ;  $df=11$ ;  $p=0.012$ ) within the left brain damaged group.

It is conceivable that the facial movement disorder of dysphasic patients contributes to the reduced spontaneous facial expression found in left lesioned patients during card sorting. However no difference in spontaneous facial movement was found when dysphasics and left hemisphere non-dysphasics were compared ( $U=28.5$ ;  $p=0.76$ ). Furthermore, in the left hemisphere group spontaneous facial movement and imitation of facial movement were not significantly correlated (Table 30). Upper face speech markers provided another index of non-emotional facial movement. Though this measure was also unrelated to spontaneous facial movement in the failure situation (Table 30), there is doubt about the validity of this latter result as it was not always possible to reliably assess upper face speech markers in a few dysphasic patients with moderate word finding difficulties and/or a non-fluent dysphasia.

	<u>Right</u> <u>Hemisphere</u>	<u>Left</u> <u>Hemisphere</u>
Upper face movements in speech	0.21	0.22
Imitation of face movement	-0.22	0.15
Verbal IQ	-0.09	0.42
Performance IQ	-0.10	0.03
Memory		
Faces	-0.20	-0.18
Words	0.14	0.32
Token Test	-	0.20
Neglect		
Cancellation	0.29	-
Bisection	0.12	-

Table 30

Correlation between measures of general sensori-motor deficit and spontaneous facial movement in the hemispheric groups.

Spontaneous facial movement in the left hemisphere group was not only unrelated to the two measures of facial movement, it was also unrelated to the several indices of cognitive function employed in the present study (Table 30).

**Right hemisphere cognitive-motor deficit:** It is not certain that special motor control functions are lateralised in the right hemisphere. A general hypokinesia and increased reaction time has been linked with right brain damage (Valenstein and Heilman, 1981; Howes and Boller, 1975), but reaction time also commonly increases after unilateral left hemisphere lesions (e.g. Dee and Van Allen, 1972). Expressive amusia has been associated with right hemisphere damage (McFarland and Fortin, 1982), but again expressive amusia can occur



after unilateral left hemisphere lesions (Brust, 1980). Lastly, it has been shown that modulation of intonation during speech can be impaired after right hemisphere damage (Weintraub *et al.*, 1981), whereas after left hemisphere lesions speech intonation may be preserved in some patients with fluent dysphasia. However speech can lack melodic contour in non-fluent dysphasics (Goodglass and Kaplan, 1972). It has also been suggested that the right hemisphere may prove to be responsible for other prosodic features accompanying speech (Ross and Mesulam, 1979), and it was partly to investigate this possibility that upper face speech markers were quantified in the present study.

While upper face speech markers were reduced after anterior damage (See Chapter 5), there was no indication that the hemispheric groups differed for upper face movements accompanying speech ( $t=1.24$ ;  $p=0.23$ ). Furthermore, within the right hemisphere group, upper face movements during speech and spontaneous facial movement were unrelated (Table 30). Lastly, table 30 shows that, within the right hemisphere group, no relationship could be demonstrated between reduced spontaneous facial movement and imitation of facial movement sequences, or indices of perceptual-cognitive deficit.

#### Lateralisation of emotional functions and other indices of reaction

**Asymmetries of facial expression:** The present finding that amount of spontaneous facial reaction was equivalent in the right and left lesion groups appears to go against the view that emotional disorder is related to side of lesion. However patient's emotional reactions to failure are not totally defined by a frequency count of facial action units. For instance, facial expressions can be bilateral or unilateral, and several studies of non-brain damaged subjects have found left unilateral facial expressions to be more common, and the left

hemiface to express emotion more intensely (Moscovitch and Olds, 1982; Borod and Caron, 1980; Campbell, 1978).

It has been concluded that the bias for left unilateral hemifacial expression indicates right hemisphere emotional dominance, and Bruyer (1981) supported this conclusion when he investigated patients with unilateral cerebral lesions. This hypothesis predicts that fewer left unilateral facial expressions would be exhibited after right hemisphere disease. Conversely, the bias towards the production of left unilateral facial expressions should be preserved in the left lesion group.

However, right and left lesion groups did not differ in the proportion of left-to-right unilateral facial expressions (Fisher Exact  $p=0.95$ ). The inference that the bias for left unilateral expressions indicates emotional dominance of the right cerebral hemisphere can also be questioned as left unilateral facial movements were more common than right unilateral facial movements in the brain damaged group as a whole (Binomial test, two tailed  $p=0.004$ ). A bias for left unilateral facial expressions even after **right** hemisphere lesions could indicate that asymmetries of facial expression are due to lateralised processes at the subcortical or brain-stem levels rather than to functional asymmetries of the cerebral hemispheres. It is also problematic that no bias to left unilateral facial movement was found in the control group, and in any case, after reviewing the literature Thompson (1985) concluded "There appears to be little consensus indicating that one side of the face is more expressive in the production of facial emotions."

**Qualitative measures of emotion:** The loss of information about unilateral facial expressions is only one of the costs incurred following the reduction of complex emotional reactions into a simple frequency count of facial

movements. Only one aspect of the patient's emotional response is represented by amount of facial motion. To correct this deficiency the patient's behaviour after each card sort was given a qualitative rating for the degree and type of emotion expressed.

Mean qualitative ratings were significantly different in right hemisphere, left hemisphere, and control patients for reactions after incorrect card sortings (Table 31). Comparing hemispheric groups, post hoc Scheffe tests showed negative reactions to incorrect sortings were significantly more pronounced in the left hemisphere group, and there was a tendency for more marked positive reactions after left sided damage ( $U=73.5$ ;  $p=0.07$ ). Therefore though the hemispheric groups were equivalent for mean spontaneous FACS action units, qualitative impressions indicated more intense emotional reactions in the left lesioned group. A closer relationship between quantitative and qualitative ratings had been predicted, and we must ask whether either or both of the quantitative or the qualitative measures provide an inadequate index of emotion.

The quantitative index of facial movement used the established Facial Action Coding System of Ekman and Friesen (1978). Furthermore, using a frequency count of facial movement Kolb and Milner (1981b) also found no differences between right and left hemisphere groups during card sorting. In the case of the qualitative index, though it has not been extensively validated, interjudge reliability and test-retest reliability were at acceptable levels. Furthermore, the increase of qualitative negative emotional response in the present left lesioned patients is in agreement with the findings of Buck and Duffy (1980) and Gainotti (1972), who both relied on qualitative impression. Similarly, the qualitative impressions of a number of investigators (e.g. Rossi and Rosadini, 1967) suggest the catastrophic reaction is more frequent during

barbiturate induced anaethetisation of the left cerebral hemisphere. In this connection, it is important to note that Kolb and Milner (1981b) were unable to demonstrate qualitative emotional differences between hemispheric groups during the Wada test.

	<u>Right</u> <u>Hemisphere</u>	<u>Left</u> <u>Hemisphere</u>	<u>Controls</u>	<u>p</u>
<b>QUALITATIVE</b>				
<b>Correct Sort</b>				
(i) positive	0.6	1.8	0.6	0.38
(ii) negative	0.4	0.5	0.4	0.98
<b>Wrong Sort</b>				
(i) positive	1.9	3.1	5.5	0.02
(ii) negative	3.8	11.0	8.0	0.004
<b>SELF-REPORT</b>				
(i) positive	7.8	9.7	7.6	0.17
(ii) negative	6.7	8.9	7.2	0.33
<b>HEART-RATE</b>				
(i) baseline	78.3	80.7	93.4	0.05
(ii) correct	77.4	81.1	90.7	0.09
(iii) wrong	76.7	77.7	88.5	0.09

Table 31

Right hemisphere, left hemisphere, and control patients are compared on qualitative, self-report, and autonomic indices. The Kruskal-Wallis one-way analysis of variance procedure was used to compare the groups for degree of positive and negative reaction after a correct card sort, and for positive reaction after an incorrect sort. Parametric procedures were used in the remaining comparisons.

It is therefore conceivable that the different results from qualitative and quantitative measures will prove to be a robust and replicable finding. Certainly, these two indices could focus on different aspects of emotional behaviour. Quantity of facial movements may measure intensity of emotional

response. Timing and patterning of spontaneous facial movement sequences is also crucial to communication of affect, and these latter features were picked up in the qualitative, but not in the quantitative assessment. Finally, the count of FACS action units depends solely on facial expression, but facial movement is only one of the guides for a qualitative evaluation of emotional reaction. The emotional content and tone of the patient's comments were taken into account for the qualitative rating. Head movements can also communicate emotion, and these were significantly more frequent in the left lesion group (Table 29).

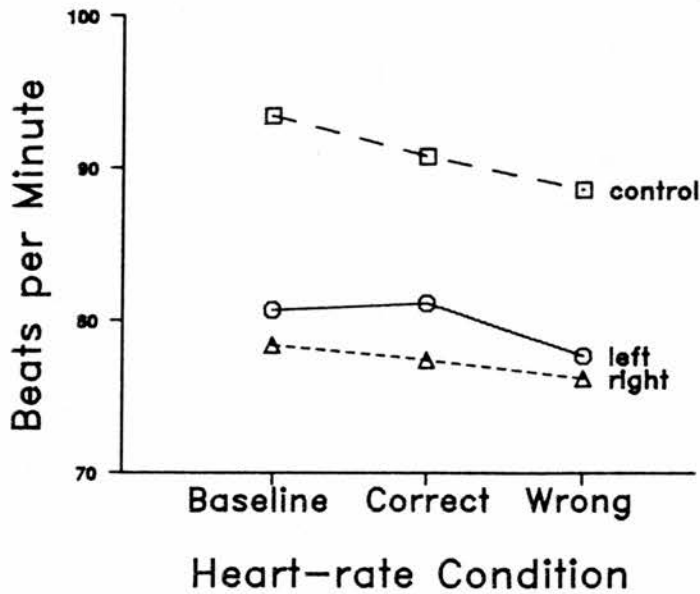


Figure 17

Heart-rate of control, left hemisphere and right hemisphere groups during baseline, correct, and incorrect sortings.

**Self-report:** A self-report measure of subjective response was also used to gain a more comprehensive assessment of emotional response. The strongest positive and negative reactions occurred in the left lesion group (Table 31), but these hemispheric differences were not significant for positive ( $U=97.5$ ;  $p=0.28$ ), or negative ( $t=1.56$ ;  $df=30$ ;  $p=0.13$ ) subjective reaction. In a more detailed analysis, each of the 10 adjectives which made up the self-report measure was considered separately, but no difference between the hemispheric groups was found for any individual adjective ratings.

**Heart-rate:** In a repeated measures analysis of variance, lesion group (right v left v control) was the between subjects factor, with repeated measures for heart rate condition (baseline v correct v wrong). The lesion group effect approached significance [ $F(2,27)=3.27$ ;  $p=0.055$ ]. Post hoc statistical testing showed that this was due to the 7 controls having higher mean heart-rate than either of the brain damaged groups, while the 14 right and the 9 left hemisphere lesion patients did not differ on any heart-rate measure. The effect of heart-rate condition achieved significance [Wilks Lambda=0.7391; Approximate  $F(2,26)=4.59$ ;  $p=0.02$ ], with heart-rate generally highest during baseline and lowest throughout incorrect card sortings (Figure 17). This fall in heart-rate from baseline levels may be determined by factors extraneous to the emotional responses. Baseline heart-rate was measured first, a few minutes after the patient had travelled a short way through the hospital to get to the room where expressive behaviour was filmed. Presumably, heart-rate would be highest just after this short journey. Over the next 15 minutes the patient performed the expressive tasks in a seated position, and this less active body state is a likely cause of the drop in heart-rate. The fall in heart-rate would be most noticeable during the card sorting task, which was administered last. Figure 17 shows that this fall in heart-rate was fairly similar in each of the 3

lesion groups, therefore a significant interaction between lesion group and heart-rate condition was neither expected nor found [Wilks Lambda=0.8883; Approximate  $F(4,52)=0.79$ ;  $p=0.54$ ].

	<u>Left</u> <u>Dysphasic</u>	<u>Left non-</u> <u>Dysphasic</u>	<u>Right</u> <u>Hemisphere</u>	<u>p</u>
<b>QUALITATIVE</b>				
<b>Correct Sort</b>				
(i) positive	2.1	1.3	0.6	0.38
(ii) negative	0.6	0.4	0.4	0.98
<b>Wrong Sort</b>				
(i) positive	1.7	3.9	1.9	0.14
(ii) negative	13.4	9.1	3.8	0.006
<b>SELF-REPORT</b>				
(i) positive	10.3	9.1	7.8	0.33
(ii) negative	12.1	5.6	6.7	0.003
<b>HEART-RATE</b>				
(i) baseline	74.2	85.0	78.4	0.31
(ii) correct	74.0	85.4	77.4	0.25
(iii) wrong	74.1	79.5	76.7	0.70

Table 32

Dysphasic, left hemisphere non-dysphasic, and right hemisphere patients are compared on qualitative, self-report, and autonomic indices, using the Kruskal-Wallis one-way analysis of variance procedure. Negative emotional response is particularly high in the dysphasic group on self-report and the qualitative rating after incorrect card sortings.

We have so far concluded that the emotional reactions of right and left hemisphere groups were not distinguishable using a frequency count of facial action units, but other emotional indices indicated hemispheric differences. In the main, these differences did not achieve significance. However it is worth noting that, where the hemispheric differences attained significance, it was the left hemispheric patients who showed the most intense expression of emotion.

Furthermore, in the remaining hemispheric comparisons, mean scores were lowest in the right lesion group. We must next interpret these differences in the light of current theories.

**Emotional reactions and dysphasia:** In two seminal studies Gainotti (1972 & 1969) found patients with left hemisphere disease were particularly prone to display aggressive or anxiety reactions when confronted with difficult neuropsychological test items. Similarly, in the present study, qualitative ratings of negative emotional responses were highest in the left hemisphere group. The present data also supported Gainotti's (1972) further observation that these emotional reactions were particularly common in patients with dysphasia.

Left hemisphere patients with dysphasia were compared with left non-dysphasic and right hemisphere patients on the qualitative, self-report, and heart-rate indices of emotional reaction. The group effect was significant for two measures of negative emotional response, and in both instances, mean negative reaction was highest in the dysphasic group (Table 32). On the qualitative index of negative reaction the difference between dysphasics and right lesion patients was significant ( $U=9.5$ ;  $p=0.0008$ ), but dysphasic and non-dysphasic left hemisphere patients did not differ significantly ( $U=20$ ;  $p=0.25$ ). Nevertheless, for the self-report measure of negative reaction post hoc testing showed dysphasic patients reported significantly more negative subjective reaction than either right hemisphere patients ( $U=13$ ;  $p=0.001$ ), or left non-dysphasic subjects ( $U=2.5$ ;  $p=0.001$ ). Furthermore, when ratings of dysphasics and non-dysphasic patients were compared for each of the 10 adjectives of the self-report questionnaire, dysphasics rated themselves as significantly more irritated ( $U=8$ ;  $p=0.021$ ), anxious ( $U=4.5$ ;  $p=0.014$ ), and they tended to feel more upset ( $U=11$ ;  $p=0.07$ ). This supports Gainotti's original observation of a catastrophic reaction which is (a) composed of aggression and



anxiety responses, and (b) associated with dysphasia. [Gainotti (1972) also found qualitative differences in the emotional reactions of fluent and non-fluent dysphasics. Initial efforts at making this classification were abandoned. There was limited time for testing, the investigator was reluctant to ask patients with severe dysphasia to engage in the 5 minutes of free speech required for assessing fluency (Goodglass and Kaplan, 1972), and in any case, non-fluent dysphasia may be relatively uncommon in patients with cerebral tumour (Coughlan and Warrington, 1978).]

Warrington (1975) has suggested that left hemisphere sites are specialised for the process of semantic categorisation, and dysphasic patients have special problems with the Weigl sorting test (De Renzi *et al*, 1966). It is likely that at least some dysphasics lacked the conceptual flexibility required for the present card sorting task, and their special cognitive difficulties may have made them particularly prone to become irritable and anxious. However the administration procedure for the card sorting test was designed with the intention of making it equally difficult for all patients. To avoid the frustrations of anomia a verbal response was not required, and patients could indicate the appropriate sorting position by pointing. Also, clues were given when patients found card sorting particularly difficult, or sorting rules were made more complicated when patients found the task too easy. By this means patients never made more than 12 consecutive wrong or correct sorting sequences. In the main, patients were unaware that these adjustments to task difficulty were being made, and it was always possible to ensure that half of the 40 card sorts were correct.

In spite of these precautions, we cannot be sure that the sorting task was in fact equally difficult for all patients, as dysphasics made a higher proportion of perseverative sorting errors than non-dysphasics ( $U=4$ ;  $p=0.005$ ). Analysis of covariance might have permitted a comparison between dysphasic and

non-dysphasic patients, with negative reaction scores adjusted for number of perseverative sorts. However the dysphasic and non-dysphasic regression equations between dependent variable (negative reaction scores) and covariate (perseverative errors) were not comparable. These regressions did not fall within the same score range, and perseverative scores were at near-ceiling levels in the dysphasic subgroup. Analysis of covariance was therefore inappropriate, and it remains possible that dysphasics' verbal-cognitive deficits are largely responsible for increased frustration and anxiety during card sorting.

**Emotional reaction and neglect:** Heilman *et al.* (1978) agree that failure is a necessary condition for the appearance of the catastrophic reaction, but for them, it is not a sufficient condition. In their view the left hemisphere normally exerts inhibitory control over arousal/emotional processes which are lateralised in the right hemisphere, and consequently left sided damage leads to disinhibition of right hemisphere arousal/emotional processes. This heightened arousal/emotional reactivity after left hemisphere damage is the additional necessary condition for the emergence of the catastrophic reaction, which in their opinion, is an enhanced or disinhibited emotional response to the stress of failure.

Heilman *et al.* (1978) compared the galvanic skin responses (GSR) of patients with dysphasia and patients with neglect (which, in their view, is an attentional/emotional disturbance). They found flat GSR responses in the patients with neglect, while dysphasic patients showed enhanced GSR relative to controls. Similarly, in the present study patients with neglect had lower self-report negative reaction scores than dysphasics ( $U=6.5$ ;  $p=0.035$ ), and they also tended to have a lower qualitative negative reaction ratings than dysphasics ( $U=6$ ;  $P=0.073$ ). Furthermore, in comparison with controls, self-report negative reaction scores were significantly higher in the dysphasic group

( $U=10.5$ ;  $p=0.023$ ), and dysphasics also tended to have higher qualitative ratings of negative reaction ( $U=11.5$ ;  $p=0.054$ ).

	<u>Line Cancellation</u>	<u>Line Bisection</u>
<b>QUALITATIVE</b>		
<b>Correct Sort</b>		
(i) positive	0.09	0.01
(ii) negative	0.31	-0.02
<b>Wrong Sort</b>		
(i) positive	-0.20	-0.22
(ii) negative	0.28	0.28
<b>SELF-REPORT</b>		
(i) positive	0.17	0.08
(ii) negative	0.49*	0.25
<b>HEART-RATE</b>		
(i) baseline	-0.46	-0.44
(ii) correct	-0.45	-0.42
(iii) wrong	-0.46	-0.44

Table 33

The relationship between two measures of visual neglect and the several indices of emotional reaction within the right hemisphere group. Spearman non-parametric correlation coefficients were computed for positive and negative reaction after a correct card sort, and for positive reaction after an incorrect sort. Otherwise, Pearson's correlation coefficients are reported (\* -  $p<0.05$ ).

However Morrow *et al.* (1981) only partially confirmed Heilman *et al.*'s (1978) findings, as they could not demonstrate enhanced GSR reactivity in the left brain damaged group. Similarly, in certain important respects the present data appear inconsistent with the views of Heilman and his associates. Presumably, their theory predicts emotional responses would be weakest when neglect is

most severe i.e. degree of neglect should correlate negatively with scores on the several emotional indices applied in the present study. In the event, the heart-rate indices correlated negatively with line bisection and cancellation scores, but no coefficient achieved significance (Table 33). Indeed, contrary to prediction, neglect on the line cancellation test was associated with significantly **stronger** negative subjective reaction (Table 33). Furthermore, as the line bisection and cancellation tests are not entirely reliable in identifying patients with neglect, right hemisphere patients with (N=5) and without (N=12) clinically diagnosed neglect were compared, but again no differences between the subgroups on the several emotional measures reached significance. Finally, patients with neglect were no different from controls on either the self-report measure of negative reaction ( $U=25$ ;  $p=0.86$ ), or the qualitative rating of negative response after incorrect card sorting ( $U=12$ ;  $p=0.28$ ).

**Differential hemispheric functional organisation:** Data from the test of spontaneous facial expression gave no support for the view that functions are diffusely represented in the right hemisphere, and focally organised in the left hemisphere. First, contrary to prediction, both right and left anterior subgroups made fewer spontaneous facial movements than the right and left posterior subgroups (Figure 16). Secondly, correlation coefficients between spontaneous facial movement and perceptual-cognitive-motor tests were low and non-significant in both hemispheric groups (Table 30), and there was no evidence that inter-test correlation coefficients were generally higher after right hemisphere damage.

**Differential lateralisation of emotion hypothesis:** In accordance with this hypothesis, patients with left hemisphere lesions displayed the strongest negative emotional reactions after incorrect card sortings (Table 31). However this enhanced negative response was associated with dysphasia, and it is

possible that dysphasics had more intense negative reactions because they experienced greater difficulty with the card sorting task. Furthermore, left hemisphere lesions should lead to a selective reduction of positive emotional response, as the left (affectively positive) hemisphere is now less able to inhibit right (affectively negative) processes. In fact, the opposite trend appeared, positive reaction was generally slightly higher in the left lesion group (Table 31). Indeed, this tendency for the left lesion group to display more positive response than the right lesion group approached significance after incorrect card sorts ( $U=73.5$ ;  $p=0.066$ ).

The differential lateralisation of emotion hypothesis also predicts a selective reduction of negative emotional response in the right lesion group. Certainly, during incorrect card sortings the right lesion patients displayed less intense negative emotional reactions than controls during incorrect card sortings ( $t=2.33$ ;  $df=22$ ;  $p=0.029$ ). However positive reaction was neither preserved nor enhanced in the right lesion group. Instead, contrary to the differential lateralisation of emotion hypothesis, patients with right sided lesions displayed less positive reaction than controls after incorrect card sorts ( $U=24.5$ ;  $p=0.013$ ).

Though these findings are inconsistent with the differential lateralisation of emotion hypothesis, there is some doubt about the validity of the qualitative measures of positive emotional reaction. Patients were said to display a positive response if they smiled, as it was possible to make this judgement fairly reliably. However sometimes the patient smiled because they were happy, and were enjoying themselves, while on other occasions they smiled to cover anxiety or annoyance. Though these latter smiles do not indicate a positive emotional response, it was not easy to reliably distinguish these two types of smile during piloting, and so in the main study the social-emotional meaning of the smile was not considered. It would be desirable to assess the differential

lateralisation of emotion hypothesis, using a more precise or "cleaner" measure of positive emotion.

### Conclusions

1. Though spontaneous facial movement in right and left anterior subgroups was depressed relative to both posterior subgroups, patients with right and left lesions did not differ in the amount of facial movement elicited by the failure situation, and there was no interaction between inter- and intra-hemispheric dimensions.
2. Within the right and the left hemispheric groups, scores on tests of sensori-cognitive-motor deficit were unrelated to level of spontaneous facial movement. However other putative indices of emotional reaction indicated the presence of an enhanced negative emotional response in left hemisphere dysphasic patients. There were indications that these negative reactions occur in response to special verbal-cognitive deficit.
3. Different reactions to particular cognitive deficits may not fully account for the differences between the hemispheric groups on emotional measures. While other more specifically emotional functional hemispheric asymmetries may play a part, these present data did not support the attentional/emotional hypothesis of Heilman and his associates. Also, the view that expressive functions are diffusely represented in the right hemisphere was not confirmed. Lastly, the data could not be easily explained by the differential lateralisation of emotion hypothesis of Sackheim *et al.* (1982).

### 7.2. Voluntary Expression of Emotion

Patients were asked to make smiling, angry, surprised, and disgusted facial expressions. Total number of FACS action units produced in all 4 expressions was made the dependent variable in a 3x2 analysis of variance; with lesion group (right v left v control), and sex as factors. The lesion group main effect was not significant [ $F(2,33)=1.43$ ;  $p=0.25$ ]. As the sex main effect [ $F(1,33)=1.09$ ;

$p=0.30$ ], and the sex by hemisphere interaction [ $F(2,33)=1.68$ ;  $p=0.20$ ] were not significant, the 20 males and the 19 females were considered together in all subsequent analyses.

<u>Action Units</u>	<u>Right Hemisphere</u>	<u>Left Hemisphere</u>	<u>Controls</u>
19-20		1	
18-17		1	1
16-15	2	4	
14-13	6	2	3
12-11	2	4	4
10-9	4	1	1
8-7	1	1	
6-5	1		

Table 34

Total number of FACS action units produced by hemispheric and control groups in the test of voluntary expressed emotion.

Total FACS action units produced by the 9 controls, 16 right hemisphere and 14 left hemisphere patients are given in Table 34. Upper and lower face movements were considered separately using the Kruskal-Wallis one-way analysis of variance procedure, but the three lesion groups did not differ for upper face ( $X^2=2.64$ ;  $p=0.27$ ), or lower face ( $X^2=2.03$ ;  $p=0.36$ ) movements.

#### Voluntary emotional expression with lesion size, type and location

To control for the comparatively minor differences between the hemispheric groups in lesion size and type, all metastases and arteriovenous malformations were excluded (3 right and 3 left hemisphere), and total lesion size was partialled out in a one-way analysis of covariance. However there was still no

significant difference between right and left hemisphere groups for total facial movements [ $F(1,21)=1.66$ ;  $p=0.21$ ].

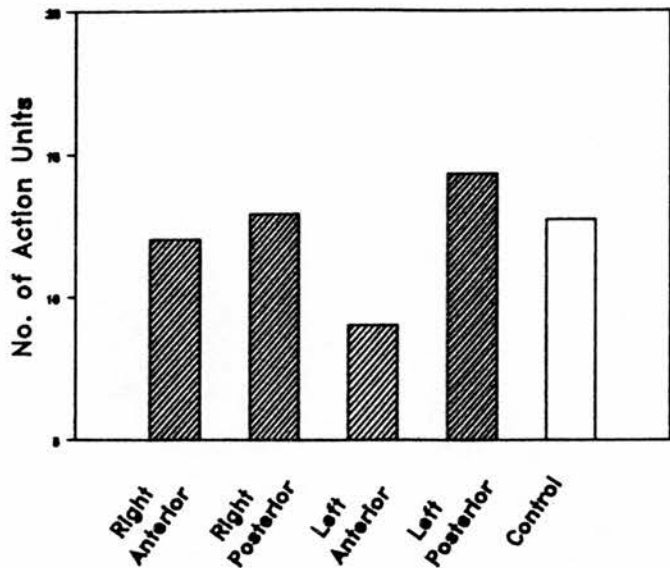


Figure 18

Mean number of FACS action units produced by the 4 lesion subgroups and controls in the voluntary production of emotional expressions.

Figure 18 gives mean total facial movement for the right anterior ( $N=3$ ), left anterior ( $N=3$ ), right posterior ( $N=8$ ), left posterior ( $N=6$ ) subgroups and the controls. The inter- and intra-hemispheric factors were considered simultaneously in a  $2 \times 2$  analysis of variance, and the intra-hemispheric main effect was significant [ $F(1,16)=6.71$ ;  $p=0.02$ ], but the effect of lesion side was not significant [ $F(1,16)=0.01$ ;  $p=0.93$ ]. However an additive statistical model may not provide the best fit to these data, as the interaction between the inter- and intra-hemispheric factors approached significance [ $F(1,16)=3.68$ ;  $p=0.073$ ]. Post



hoc statistical testing indicated that this interaction was due to the apparently selective reduction of voluntary facial expression in the left anterior subgroup. Of course, the right anterior and left anterior subgroups each contained only 3 patients, and the significance of the interaction term did not attain the conventional level of significance. Nevertheless, at the very least these data demonstrate the value of considering the inter- and intra-hemispheric factors simultaneously, by illustrating the inadvisability of simply assuming an additive statistical model.

**Relationship between perceptuo-motor and cognitive deficit and facial movement in voluntary emotional expression**

Table 35 indicates no strong associations between voluntary facial movements and any of the measures of general sensori-cognitive-motor deficit, within either of the hemispheric groups. Voluntary emotional movement and upper face speech markers were positively related within the left hemisphere group, but this only approaches significance ( $p=0.08$ ), and imitation of facial movement was unrelated to voluntary facial movement. In the right hemisphere group neither of the measures of non-emotional facial movement correlated with voluntary facial movement. However, facial recognition memory scores were highest in the right hemisphere patients who produced the fewest facial movements in their voluntary emotional expressions. There was no other occasion when recognition memory was negatively associated with an emotional measure, and though significant, this negative association between facial memory and number of action units produced in voluntary emotional expressions is not easily explained. It is concluded that the association between voluntary emotional expression and facial recognition memory is probably spurious.

	<u>Right</u> <u>Hemisphere</u>	<u>Left</u> <u>Hemisphere</u>
Upper face movements in speech	0.22	0.51
Imitation of face movement	0.01	-0.11
Verbal IQ	-0.38	0.07
Performance IQ	0.12	-0.07
Memory		
Faces	-0.50*	-0.09
Words	0.02	-0.09
Token Test	-	0.00
Neglect		
Cancellation	-0.29	-
Bisection	-0.39	-

Table 35

Correlation between measures of general sensori-motor deficit and voluntary emotional facial movements in the hemispheric groups.  
(\* -  $p < 0.05$ )

### Lateralisation of emotional functions and voluntary emotional expressions

Sometimes patients generated significant facial movement but they failed to adequately portray the required emotion. Therefore in addition to the quantitative count of FACS action units produced, each voluntary emotional expression was given a qualitative rating of its adequacy. Within the brain damaged group as a whole, there was a significant correlation between quantitative and qualitative scores ( $r=0.73$ ;  $p<0.001$ ), and in most respects the lesion groups performed similarly on both measures. For instance, as with the quantitative index, the group effect was not significant when total qualitative

ratings for the lesion groups (right v left v control) were compared in a one-way analysis of variance [ $F(2,36)=0.93$ ;  $p=0.41$ ]. However the quantitative and qualitative indices gave different results in a  $2 \times 2$  analysis of variance when the intra- and inter-hemispheric dimensions were factors. With the **quantitative** index the interaction between intra- and inter-hemispheric factors approached significance, because left anterior patients showed a selective reduction in facial movement (Figure 18). While **qualitative** ratings were also significantly lower for anterior patients [ $F(1,16)=21.74$ ;  $p<0.0001$ ], the interaction between inter- and intra-hemispheric factors was not significant [ $F(1,16)=0.37$ ;  $p=0.55$ ], as right and left anterior groups were equally impaired (Figure 19).

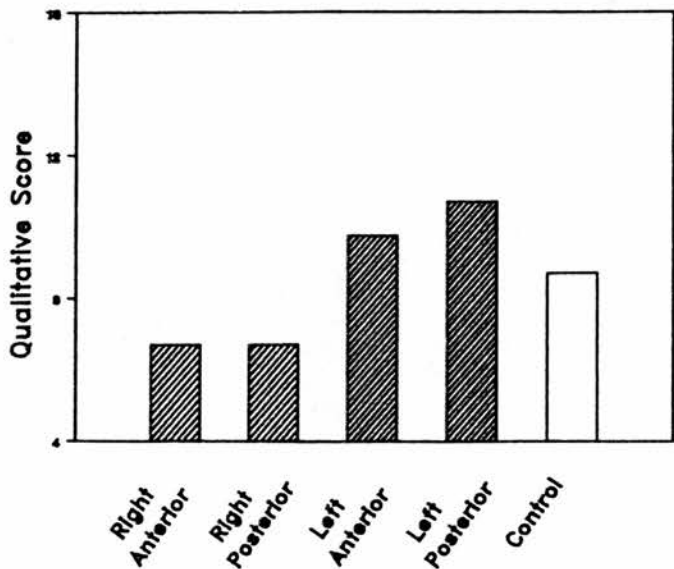


Figure 19

Mean qualitative ratings of the 4 lesion subgroups and controls for voluntary emotional expressions.

Of course, this apparent dissociation between quantitative and qualitative performance may be spurious, as subgroup sizes were low, and for most comparisons similar conclusions were derived from quantitative and qualitative scores. Nevertheless, it is unwise to exclude the possibility that the two indices measure different aspects of voluntary emotional expressions.

**Voluntary emotional expression and dysphasia:** As with quantitative scores, qualitative ratings were not significantly associated with Token Test score ( $r=0.22$ ;  $df=10$ ;  $p=0.50$ ). Furthermore, mean qualitative ratings for the 8 dysphasic patients were no different from mean ratings of either the 7 non-dysphasic left hemisphere subjects ( $U=22.5$ ;  $p=0.54$ ), or the 16 right hemisphere subjects ( $U=52$ ;  $p=0.82$ ).

This contrasts with spontaneous emotional reaction in the card sorting situation, where dysphasic patients had higher qualitative ratings of negative emotional reaction. Probably the simplest way of accounting for this difference is to suggest that dysphasic patients displayed increased negative reaction because the card sorting task was particularly difficult for them, but dysphasics did not have special problems with the voluntary emotional expression task because the test instructions were easily understood. Indeed, if some of the present patients had had a severe global dysphasia, and had not understood instructions for the test of voluntary emotional expression, dysphasia might have been associated with lower (rather than higher) qualitative ratings.

**Voluntary emotional expression and neglect:** The attentional/emotional hypothesis of Heilman and his associates was not supported. First, though mean qualitative and quantitative scores were lower in the right hemisphere group, the right-left differences were not significant. Indeed, Heilman *et al.* (1983) themselves assessed voluntary emotional expression after unilateral

brain damage, but found no difference between right and left lesion groups. However Bruyer (1981) did find voluntary emotional expressions were less intense after right hemisphere damage. Secondly, qualitative ratings were unrelated to line bisection scores ( $r=-0.33$ ;  $df=13$ ;  $p=0.24$ ), and line cancellation scores ( $r=-0.28$ ;  $df=13$ ;  $p=0.33$ ). Furthermore, no significant difference was found between patients with ( $N=5$ ) and without ( $N=12$ ) neglect on the qualitative rating ( $U=16$ ;  $p=0.16$ ), or quantitative score ( $U=16.5$ ;  $p=0.17$ ). Thirdly, Heilman and his associates have sometimes attempted to control for degree of perceptual-cognitive deficit by comparing left hemisphere dysphasic patients and right lesioned patients with neglect. However these groups did not differ significantly on either the quantitative measure ( $U=11$ ;  $p=0.34$ ), or qualitative index ( $U=10$ ;  $p=0.27$ ).

**Differential hemispheric functional organisation:** There was no evidence to suggest that the functions involved in the voluntary production of emotional expressions were more diffusely represented in the right hemisphere. First, table 35 shows that in the hemispheric groups correlation coefficients between sensori-motor measures and voluntary facial movement were comparable in magnitude. Secondly, contrary to the view that functions are differently organised in the each hemisphere, a similar anterior-posterior difference was found for patients with right and left hemisphere lesions on qualitative ratings (Figure 19). Of course, quantitative scores were significantly lower in the left anterior group in comparison with left posterior patients, while right anterior and right posterior groups did not differ significantly (Figure 18). However this does not necessarily support the focal/diffuse dichotomy, as neither right anterior nor right posterior subgroups were impaired (relative to controls) on the quantitative index.

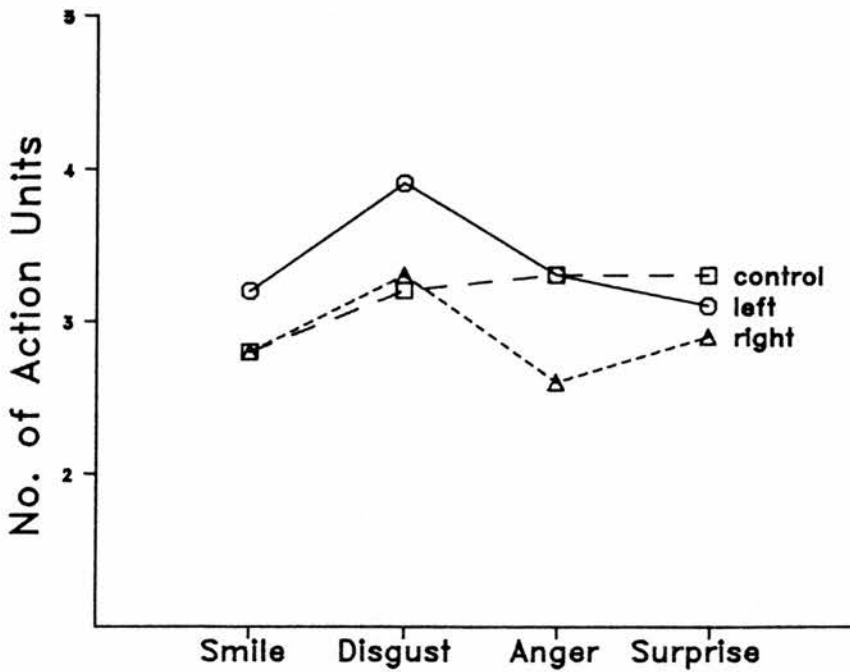


Figure 20

Mean action units produced by right hemisphere, left hemisphere, and control groups in the voluntary expression of happy, angry, disgusted, and surprised feeling.

**Differential lateralisation of emotion hypothesis:** Quantitative and qualitative scores for the 4 emotional expressions were compared in the 3 lesion groups using the Kruskal-Wallis one-way analysis of variance procedure. As findings were identical for both measures, only the results using the quantitative index are reported. The group effect was not significant for smiling ( $X^2=0.40$ ;  $p=0.82$ ), or angry ( $X^2=3.61$ ;  $p=0.16$ ), or disgusted ( $X^2=2.02$ ;  $p=0.36$ ), or surprised ( $X^2=0.33$ ;  $p=0.85$ ) expressions. Furthermore, the differential lateralisation of emotion hypothesis predicts that the ability to smile would be relatively preserved in patients with right hemisphere disease, while capacity for negative emotional

expression would be spared in patients with left hemisphere damage. Figure 20 shows that neither prediction was confirmed.

### Conclusions

1. Qualitative ratings of voluntary emotional expression were depressed in both anterior subgroups, but the difference between the right and left hemisphere groups was not significant. However on the quantitative index, significant reduction of facial movement occurred only in the left anterior group. This difference between qualitative and quantitative scores may be unreliable, because in all other comparisons quantitative and qualitative scores gave identical results.
2. Within both hemispheric groups neither quantitative nor qualitative indices were significantly associated with scores on tests of sensori-cognitive-motor deficit. In particular, there was no association between dysphasia and voluntary emotional expression scores.
3. These data did not support the attentional/emotional hypothesis, the focal/diffuse dichotomy, or the differential lateralisation of emotion hypothesis.

### 7.3. Discussion

The use of multiple measures suggests a complex relationship between emotional disorder and lesion site. In particular, a focal lesion may lead to low scores on one index of emotion without inducing corresponding reductions on other emotional measures, and certainly table 20 shows that correlations between various indices of emotional functioning were generally rather low. Of course, dissociations between different emotional indices are normal in brain damaged subjects (Morrow and Labrun, 1978), and emotional indices were not highly inter-related in the present non-brain damaged control group. Nevertheless, Ross and Rush (1981) suggested that differently located lesions

can be shown to selectively disturb individual components of emotional behaviour. Indeed, during card sorting in the present study emotional indices were sometimes differently related to lesion site. Patients with anterior lesions had lower scores than patients with posterior lesions on both quantitative and qualitative measures. In contrast, side of lesion did not equally affect quantitative and qualitative scores. Thus an equal number of FACS action units was elicited in the hemispheric groups, whereas the qualitative rating of negative emotional reaction was significantly higher for patients with left hemisphere lesions.

Though the self-report measure did not evaluate exactly the same emotional events, the patients' ratings of their subjective reaction strengthened the case for dissociations between components of emotional responses following brain damage. Left hemisphere dysphasic patients reported increased irritation, upset, and anxiety, and this confirms the hemispheric group differences on the qualitative index. In contrast, when anterior and posterior groups were compared on the qualitative and quantitative measures, both indicated a general reduction in emotional response after anterior lesions. However, the self-report measure gave evidence of increased subjective emotional reaction. Though patients with anterior lesions did report less confusion, they were also more upset, and more inclined to describe the task as being childish.

Greater use of multiple measures is required in future research if (a) we are to establish the reliability of such dissociations between emotional indices, and (b) we are to define their individual relationships with lesion site. However at this early stage it is perhaps most appropriate to theoretically outline three possible ways in which the intra- and inter-hemispheric factors may combine to produce the dissociations between measures of emotional reaction. First, it



is possible that anterior cortical-subcortical systems are specialised for emotion, but emotional processes are not lateralised. Secondly, anterior systems may be specialised for some emotional processes, and some emotional functions may be lateralised. Thirdly, anterior neural systems may not be bilaterally specialised for emotion, but emotional processes are lateralised.

**1. Anterior neural systems are specialised for emotion, but emotional processes are not lateralised:** The present data are, perhaps, most consistent with this possibility. The anterior lesion group showed some deficit on all 4 of the main emotional tests employed in this study. Of course, this does not imply that there is a single frontal cortical-subcortical neural mechanism controlling all aspects of emotional behaviour. There is good evidence for anatomical and functional heterogeneity in frontal cortical-subcortical systems (Rosenkilde, 1979; Rosvold, 1972), and there is evidence that different emotional responses are controlled by partially distinguishable anatomical pathways (Siegel, 1982; Jurgens, 1979). Furthermore, one explanation for the marked dissociations between emotional test scores in subjects with or without brain damage is that emotional functions are regulated by several (at least partially) distinguishable neural systems.

If emotional processes are not lateralised, then hemispheric differences on emotional measures are presumably caused by perceptual-cognitive-motor hemispheric asymmetries. Scores on measures of emotional perception, expression, and subjective experience can be influenced by sensori-motor deficit in two general ways. First, the patient can react to the sensori-motor deficit. For example, the enhanced negative response in the left hemisphere group during card sorting, may have been caused by the peculiar cognitive handicap suffered by many dysphasic patients. Perhaps patients with right

hemisphere disease are less stressed because deficits, such as visual neglect, cause the patient less intense frustration and create a less acute sense of social isolation. Secondly, we have already discussed in some detail the way in which deficit on an emotional test can be a secondary manifestation of a primary sensori-motor disorder. For instance, the deficits of facial perception associated with right hemisphere disease are (at least partially) responsible for the particular difficulties shown by the right lesioned patients on emotional face-face matching.

**2. Anterior neural systems are specialised for some emotional processes, and some emotional functions are lateralised:** The view that emotional processes are lateralised currently takes two forms. First, it may be that some emotional functions are represented in the right hemisphere while other emotional functions are lateralised in the left hemisphere. The present data provided little support for this, and furthermore, the version of the differential lateralisation of emotion hypothesis advocated by Sackheim *et al.* (1982) was contradicted. Nevertheless, it remains possible that with other tests, the hemispheres will be shown to be specialised for different emotional functions. A second hypothesis postulates that the right hemisphere may be dominant for all emotional processes. There was a general tendency for mean scores on the emotional tests to be lower in the right hemisphere group. This tendency often did not achieve significance, but on a few occasions it was possible to argue that cognitive factors may not have been the sole cause of the greater deficit in the right hemisphere group. In spite of this none of the current theories of right hemisphere emotional dominance provided a satisfactory explanation for patient performance on the 4 main emotional tests.

The hypothesis of a special attentional/emotional function lateralised in the right hemisphere was well able to explain patient performance on the tests of

emotional perception. However it did not adequately account for the data from the tests of emotional expression. The views of Gardner and his associates were partially supported by the present data on emotional perception. However their model postulates a disturbance of emotional structure after right hemisphere disease, and this claim was not confirmed. Finally, the concept of a focal/diffuse dichotomy between the two hemispheres was partially supported by the data on emotional perception, but contradicted by results from emotional expression.

Most recent research has aimed at establishing the existence of hemispheric emotional asymmetries. Far less attention has been spent on evaluating the relationship between the putative frontal, and lateralised emotional systems. There is a clear need to differentiate those emotional functions which are lateralised from those which are mediated by frontal systems. The present data did not enable us to resolve this question. Furthermore, the possibility of "new" or "emergent" emotional functions resulting from interactions between lateralised and frontal emotional systems has only occasionally been discussed (Bear, 1983; Pribram, 1981). Though no firm evidence for such interactions was found in the present study, there were weak indications that perception of verbally expressed emotion and production of voluntary emotional expression may be especially impaired after left anterior damage.

**3. Anterior neural systems are not bilaterally specialised for emotion, but emotional processes are lateralised:** There is widespread acceptance of the view that the frontal lobes are specialised for the regulation of, at least some, emotional functions. However in some models of the neural substrate of emotion a special role for both frontal lobes is not emphasised or discussed. For instance, Ross (1981) argues that the right hemisphere is dominant for

emotional functions in the same way as the left hemisphere is dominant for language. Furthermore, right hemisphere damage produces a range of "aprosodias" which are analogous to the aphasias of the left hemisphere. Patients with left anterior lesions sometimes have a motor aphasia, where expressive speech is greatly reduced while comprehension is relatively spared. Similarly, patients with right anterior damage can display motor aprosodia which is an emotional disorder, where expression of emotion is reduced and flattened, while perception of emotion is relatively preserved. Conversely, sensory aphasia and the analogous sensory aprosodia are associated with posterior left and right hemisphere lesions, respectively. In sensory aprosodia emotional comprehension is impaired and, while emotional expression is fluent, it is sometimes inappropriate.

In the present study the greatest deficit was found in patients with right and left frontal lesions. This is inconsistent with the view that the neural substrate of emotion is distributed along the inter-hemispheric dimension, but not along the intra-hemispheric dimension.

## CHAPTER 8

### CLASSIFICATION AND ASSESSMENT OF EMOTIONAL DEFICIT AFTER BRAIN DAMAGE

Comparative lesion, neuroanatomical, and electrophysiological studies have provided new insights into neural processes. Though there are many obstacles to the integration of human and animal data (Passingham and Ettlinger, 1974; Drewe *et al.*, 1970), links between these two research domains have developed rapidly in recent years. This trend is followed in the present investigation, which used both animal and human data to formulate a method for classifying and assessing emotional deficits consequent to brain damage. The limitations of this classification system and the methods derived from it should now be discussed, in the light of the experimental evidence.

#### Multiple emotional subsystems

**Ethological classification:** We might group together functionally related behaviours. For example, in the rat the class of maternal behaviours includes nest building and retrieval of infants (Jacobson *et al.*, 1980). This category would be distinguishable from the class of offensive-aggressive behaviours which involve threat and attack (Adams, 1979). If different neural systems control each category of behaviour we might find lesions in site A would selectively disturb maternal behaviours while sparing all offensive-aggressive behaviours; whereas lesions in site B would disrupt aggression, but not maternal behaviour.

In the present study emotions were distinguished, and we sought evidence for selective impairment in the perception or expression of particular emotion

types. On tests of emotional perception frontal patients had equal difficulty in the identification of all types of emotion. Similarly, on the tests of emotional expression both positive and negative emotional responses were generally lower in the anterior lesion group. Furthermore, the view that the right hemisphere was specialised for negative emotion, and the left for positive emotion, was generally contradicted. Of course, the general impairment of all types of emotional behaviour in the present anterior group may be due to all patients having lesions large enough to disturb several emotional subsystems simultaneously.

Though the present findings do not support the view that each category of functionally related emotional behaviours is controlled by a specific neural circuit, this experimental study was not primarily designed to investigate the ethological-functional approach to classification. The ethological method depends on observation of emotional behaviour as it occurs in natural settings. After careful observation of varied social and emotional events, these behaviours can be grouped into functional categories such as maternal behaviour, or aggressive-defensive behaviour.

The relevance of ethological categories to the study of the neural substrate of emotion is a subject of current debate (Isaacson, 1979). In the first place, the value of ethological-functional classification remains to be demonstrated empirically. Smith and Flynn (1980a&b) found different subcortical and brain stem sites were involved in the control of quiet biting and attack, but the neural substrate for the two types of aggression were only partially distinguishable anatomically. Distinct neurochemical systems have been identified, and there is good evidence that these each make an individual contribution to behavioural processes (Iversen and Iversen, 1981). However the precise contribution of these separate systems to the regulation of

ethologically defined behaviours is unclear. Using electrical brain stimulation, Roberts (1969) argues that anatomically overlapping, but separate, hypothalamic neural circuits regulate different types of emotional behaviour. However Valenstein *et al.* (1970) dispute this, and suggest the hypothalamus has a more non-specific influence on emotional behaviour, by modulating general mood and motivational states. In the second place, there are serious definitional problems for the ethological classification of emotional behaviour (Gray, 1982b; Panksepp, 1979). For instance, it was suggested that the class of maternal behaviours was distinct from the class of offensive-aggressive behaviours. How then are we to categorise the maternal aggression exhibited when the mother protects its infants? Is it offensive-aggressive, defensive-aggressive, or is it maternal-aggressive (Brain, 1979)?

**Selective impairment of components of emotion:** The present study was primarily aimed at assessing an alternative way of classifying emotional disorder. Emotional perception, emotional expression, subjective experience, and autonomic reactivity were each assessed separately. There were indications that these different components of emotional behaviour are controlled by differently located neural circuits. Thus individual emotional indices were often weakly correlated in both brain damaged (Table 20), and non-brain damaged groups. In particular, the low correlation between motoric and autonomic indices may indicate that these two components of emotional response are regulated by separate neural systems which are only weakly coupled during the production of an emotional response. These dissociations between components of emotional response were sometimes increased after brain damage. For instance, in the anterior lesion group, quantitative and qualitative indices indicated **decreased** spontaneous emotional reaction, while self-report ratings indicated a **greater** feeling of emotional upset, and a **greater** feeling that the

task was childish. In fact, Franzen and Myers (1973) also noted poverty of facial expression, together with signs of increased social anxiety after frontal lobectomy in rhesus monkeys.

Clearly, assessment of the components of emotional response has suggested that cerebral lesions can selectively disturb component emotional processes. However, much greater use of multiple measures is required, before their ability to distinguish different categories of emotional disorder is established. These difficulties in determining an appropriate method of classifying emotional disorder are compounded by an additional problem. Cognition and emotion are intimately linked, and the nature of this relationship has implications for the classification of emotional disorders. This relationship was examined in some detail in the present study.

#### Emotional impairment and sensori-motor deficits

The fundamental continuity between sensori-motor and emotional processes was discussed. Neuroanatomical studies indicate that sensory data passes through primary sensory cortex to unimodal association cortex, and then to multimodal cortex (Jones and Powell, 1970). We know most about the processing of visual sensory input. Neurophysiological investigations show that primary visual cortical neurons detect relatively simple features of the stimulus (Hubel and Wiesel, 1968), while at a late stage of visual processing, single units in association cortex respond selectively to whole objects such as faces (Perret *et al.*, 1982). It was hypothesised that when a lesion disrupts this sensory/perceptual phase, the deficit in emotional perception is secondary to the primary sensory/perceptual deficit.



The limbic system receives sensory information in highly processed form i.e. at the later stages of sensory processing (Turner *et al.*, 1980; Chavis and Pandya, 1976). Thus Thorpe *et al.* (1983) identified a set of orbitofrontal neurons which responded to a combination of visual and "limbic" information. These neurons fired on visual presentation of motivationally significant food objects such as a raisin or a banana. Convergence of limbic and highly processed sensory information on single neurons of this type may be essential for the creation of emotional perceptions. For example, a face may be perceived as threatening because visual information about the face and limbic information about anxiety/threat converge on a particular network of orbitofrontal neurons. Conceivably, damage to this neuronal network would impair capacity to perceive threatening faces, without affecting perception of the physical (non-emotional) aspects of the face, that are analysed in unimodal and multimodal sensory cortical-subcortical systems. Such a patient would perceive all aspects of the face as before, except that he would be unable to experience the face as threatening. This impairment of specifically emotional perception would fit the criteria for a pure emotional disorder.

Thorpe *et al.* (1983) identified a further set of orbitofrontal neurons which responded selectively to complete objects, as well as to the motivational value of the stimulus. The response of these neurons to the stimulus object depended on whether that object was rewarded in a learning task. Thorpe *et al.* suggested that these sensory-limbic "interface" neurons were involved in memory processes. Certainly, it is well known that "limbic" and associated structures are centrally involved in cognitive processes (Aggleton and Mishkin, 1983; Oscar-Berman, 1975; Milner, 1968; Douglas and Pribram, 1966; Williams and Pennybacker, 1954). Also, damage to the limbic and associated structures often produces a combination of memory deficits and emotional disturbance

(Damasio *et al.*, 1985). "Limbic" cognitive and emotional functions may be related in two ways. Cognitive and pure emotional functions may simply co-exist, with separate but anatomically overlapping circuits performing each role.

It is conceivable that in some cases the link between cognition and emotion is more intimate, with the same neural circuit participating in both cognitive and emotional processes. This raises the possibility that there is a class of deficits which are irreducibly cognitive-emotional. For example, patients with frontal damage can display perseverative behaviour during card sorting, as well as disinhibition of sexual and aggressive impulses. The perseveration in the cognitive task and the disinhibition of emotional responses may be separate manifestations of a unitary cognitive-emotional disorder of response control.

To investigate these three possible categories of emotional disorder it was suggested that cognitive function and emotional behaviour should be assessed together, as far as is possible. We must now evaluate the efficacy of this experimental strategy.

**Emotional deficit secondary to cognitive deficit:** The data of this study strongly indicated that perceptual-cognitive deficit was, at least partially, responsible for impaired performance on tests of emotional perception. Thus patients with visual-perceptual disturbances showed selective impairment on the subtests using facial emotional stimuli, while dysphasic patients had particular difficulty with verbal emotional material. Nevertheless, emotional deficits were also dissociable from cognitive deficits. Patients with anterior lesions generally remained significantly impaired relative to the posterior group on all 4 of the main emotional tests when the degree of sensori-motor-cognitive deficit was partially controlled. Furthermore, there were 4 posterior lesion patients who had

significant cognitive impairments, but their scores on the emotional tests was at least average for the brain damaged group.

While cognitive deficit probably cannot wholly account for impairment on the emotional measures, we must still determine the precise nature of each emotional deficit. In particular, we require operational definitions of pure emotional deficit, and cognitive-emotional deficit, if these two hypothetical categories of emotional disorder are to be distinguished.

**Pure emotional disorders:** If deficits on emotional tests in the anterior lesion group were cognitive-emotional, then the anterior-posterior difference should disappear when adjustments were made partial out cognitive deficit. However statistical controls generally did not eliminate the difference between anterior and posterior lesion groups. This tends to support the view that, in the main, pure emotional deficits were found in the frontal patients.

The case for pure emotional disorders would be further strengthened if individual cases had emotional deficits in the absence of cognitive impairment. Certainly, Mitchell-Heggs *et al.* (1976) reported preservation of cognitive function after their stereotaxic limbic leucotomy procedure for the treatment of severe psychiatric disorder. They suggested that specific limbic circuits are damaged by their small strategically placed lesions, and claim that personality was preserved while particular aberrant emotional responses were selectively suppressed. However all patients in the present sample with low scores on the tests of emotional function were also deficient on tests of general cognitive, perceptual or motor functions. Nonetheless, one patient did have low scores on 3 emotional tests (i.e. below the range for the controls and all but one of the patients with posterior lesions), while his performance was well within the normal range on most of the tests of general cognitive, perceptual, and motor

functions. However this patient did not have an unequivocally pure emotional disorder, because he showed a clear deficit on the facial recognition memory test and a possible impairment on the delayed facial identity recognition task. In any case, psychometric testing was too brief to allow us to rule out cognitive losses. Furthermore, lesion size was large in this patient, who had a frontal lobectomy. We should be surprised if he had no cognitive deficit.

**Cognitive-emotional disorders:** Having shown that when deficits on cognitive and emotional tests are dissociable, the data tend to indicate the presence of a pure emotional disorder, what can be inferred when deficits on cognitive and emotional tests are **not** dissociable? Certainly, the latter pattern of deficit would be consistent with a unitary cognitive-emotional disorder. But, deficits on both cognitive and emotional tests could also be produced by the simultaneous disruption of activity in separate but anatomically adjacent cortical-subcortical systems for (a) pure emotional functions, and (b) cognitive processes. Clearly, the an intimate association between of cognitive and emotional functions cannot be proved in an experimental design which just relies on the concurrent administration of cognitive and emotional tests.

Concurrent administration of cognitive and emotional tests could permit some limited investigation of cognitive-emotional deficit. In a cognitive-emotional disorder, emotional deficits are invariably associated with specific cognitive impairments, and a high correlation between certain emotional and cognitive scores would be predicted. For instance, after frontal lobe damage we might find particular memory deficits, or perseverative phenomena to be closely linked with certain emotional problems. Associations of this kind were not found in the present study. The significant anterior impairment on emotional tests generally remained after adjustments were made for memory deficit and perseverative errors. The attentional/emotional

hypothesis of Heilman *et al.* (1983) also falls into the category of cognitive-emotional deficit. Indeed, there was a high correlation between visual neglect and emotional perception scores, but visual neglect was not significantly related to measures of emotional expression.

To conclude, the concurrent administration of cognitive and emotional tests can help us investigate cognitive-emotional associations, but this strategy appears to be incapable of proving the existence of truly unitary cognitive-emotional deficits. Indeed, at present, it is hard to conceive of any experimental strategy capable of providing the necessary proof.

#### Emotional deficit and subcortical lesion site

The subcortical structures involved in the regulation of emotion are closely interconnected anatomically (Brodal, 1981); electrophysiological evidence indicates they are functionally linked (Rolls *et al.*, 1980); and lesions here induce emotional disorders (Isaacson, 1976). Though related, the various subcortical structures do not perform identical emotional functions. Electrical brain stimulation in different locations often elicits different emotional behaviours (Siegel *et al.*, 1972; Roberts, 1969). For instance, Jurgens (1979) found that only one type of vocalisation (chirping) could be elicited from the midline thalamus of the squirrel monkey, whereas growling calls and shrieking calls were produced on stimulation of the stria terminalis. Subcortical lesion site similarly influences the nature of emotional disorder (Numan *et al.*, 1985; Brown, 1967). For example, Kolb and Nonneman (1974) found social contact was reduced in rats after hippocampal, but not septal, lesions; while both hippocampal and septal lesions reduced shock induced aggression. The specific roles of particular subcortical structures in the production of emotional behaviour is clearly relevant to any attempt at classifying emotional deficit. The present

study allowed limited investigation of the contribution of subcortical lesion site to emotional deficit.

**Basal ganglia:** Lesions of the globus pallidus disrupt species-typical display behaviour in the squirrel monkey (MacLean, 1978). Of course, this may be the consequence of a general motor deficit, that "even handedly" impairs capacity to coordinate both emotional and non-emotional motor synergisms. Certainly, Rolls *et al.* (1979) identified neurons in the globus pallidus which responded specifically to the animal's own movements. For example, some pallidal movement-related neurons fired as long as oral movements were made, but discharge was not reward-related, since they fired equally when drinking **either** (non-rewarding) saline **or** (rewarding) glucose. These neurons contrast with the reward-related neurons in the lateral hypothalamus which responded to a visual stimulus associated with glucose, but not to a similar stimulus that had been associated with saline (Mora *et al.*, 1976).

There are claims that the basal ganglia are involved in the regulation of specifically motivational/emotional processes. In a review of the emotional disorders associated with degenerative neurological diseases of the basal ganglia Mayeux (1983) concludes, "Although...unproven, it is intriguing to consider that alterations in catechol or indolamine metabolism may underlie the emotional changes as well as motoric features of basal ganglia diseases." Furthermore, self-stimulation of neostriatal sites has been reported in rats, cats, monkeys and man (Phillips, 1979). This suggests that electrical stimulation in these sites is rewarding, which in turn implies that the neostriatum may be included within the limbic motivational-emotional circuits.

The present data did not give consistent indications about the role of the basal ganglia in the control of emotional processes. When patients with and

without basal ganglia damage were compared, the group with lesions in the basal ganglia showed significant impairment on tests of emotional perception as well as of emotional expression. This impaired performance on non-expressive emotional tasks, might suggest a special role for the corpus striatum in the regulation of emotion. However in a more rigorous comparison, patients with damage confined to a single subcortical structure were considered, and a trend for greater deficit in the basal ganglia group was evident only on the test of spontaneous facial expression. This tends to favour the hypothesis that the basal ganglia are predominantly concerned with the motor coordination of expression. Unfortunately, there were few subjects in this comparison, and it was inappropriate to use analysis of covariance to assess whether damage to the basal ganglia produced a motor disorder or an expressive deficit in specifically emotional behaviours.

**Amygdala and hippocampus:** There are strong reasons for believing that the amygdala is involved in the regulation of emotional behaviour (e.g. Kling, 1972). Less attention has been devoted to the motivational/emotional role of the hippocampus, but some have concluded it serves an emotional function (Gray, 1982a; Jarrard, 1973). It was surprising, therefore, to find that performance on emotional tests of patients with damage to medial temporal structures was equal to the performance of patients without medial temporal lesions. In particular, a deficit in perception of emotion after damage to the amygdala had been predicted, since this structure is richly supplied with highly processed sensory data (Turner *et al.*, 1980), and it has for a long time been thought to invest sensory data with emotional significance (Goddard, 1964).

**The hypothalamus:** Two patients with extrinsic lesions at the floor of the third ventricle were available for testing. One patient had a craniopharyngioma and the other had a suprasellar meningioma. Neither had significant hydrocephalus.



Both were administered the emotional tests after tumour debulking or removal, respectively. It was expected that these patients would suffer from a general impairment on all of the emotional tests, as human and animal data both strongly implicate the hypothalamus in the control of emotion. However both patients performed better than was average for the brain damaged group.

Perhaps the aetiology of the lesion accounts for the unexpected lack of emotional deficits in these two patients, whose extrinsic tumours may have produced comparatively minor hypothalamic damage. Intrinsic hypothalamic lesions (Haugh and Markesbery, 1983; Reeves and Plum, 1969) may induce more severe neuronal loss, and emotional dysfunction. However emotional symptoms have previously been reported in association with craniopharyngiomas (Killeffer and Stern, 1970; Malamud, 1967) and, in any case, there were other indications of hypothalamic damage in both patients. Postoperatively, the patient with the suprasellar meningioma suffered transient respiratory difficulties and an unusually low heart rate. She also reported increased thirst, and there were memory deficits, in the absence of impairment in IQ scores. The patient with the craniopharyngioma, had marked memory impairment, and hypersomnia, and intense thirst, and was slightly obese but did not report excessive hunger. She reported a total loss of sexual interest, without indications of an increase or a decrease in irritability. Her mood tended to be mildly positive, but this could not be described as euphoric excitement, as she often seemed to lack strong feeling. On the other hand, her behaviour did have certain manic features. She talked excessively, while her facial movements and general manner was rather exaggerated or over-expressive. Interestingly, manic behaviour has previously been described in association with lesions in this region (Malamud, 1967; Ironside, 1955). We cannot be completely satisfied that the above behavioural abnormalities were due to hypothalamic damage because this patient's husband



was quite certain that there had been no emotional changes of any kind throughout her illness.

**Dorsomedial nucleus of the thalamus:** The dorsomedial nucleus of the thalamus has close anatomical and functional links with the hypothalamus, amygdala and frontal lobes (Jones, 1981; Siegel *et al.*, 1972; Leonard, 1972; MacDonnell and Flynn, 1968). Siegel *et al.* (1972) conclude that the dorsomedial nucleus of the thalamus may have a role in the control of emotion, which is at least partially independent of the hypothalamus, and emotional symptoms have been linked with damage to this nucleus (Lobosky *et al.*, 1984; Brierley and Beck, 1958).

The contribution of this nucleus to emotion warrants more attention in the future, and in the present study subjects with suspected, or proven, damage to this nucleus were sought. Unfortunately, it was not possible to test any of the few patients with third ventricle lesions that were admitted to the wards.

#### Implications for future research

We have considered two systems for classifying the emotional disorders following brain damage. It was suggested that ethological-functional criteria may eventually provide guidelines for distinguishing emotional deficits. This approach was but briefly examined, as the present study was primarily intended to investigate an alternative (but not necessarily incompatible) mode of classification, which assumes that the components of emotional responses can be selectively disturbed after brain damage. The data obtained supported the latter approach quite well, but there were some problems. For instance, it was not possible to specify criteria to unequivocally establish the existence of the class of cognitive-emotional disorders. Furthermore, although many previous studies have indicated that the hypothalamus and medial temporal structures

are centrally involved in the control of emotional behaviour, performance on the present emotional tests was not selectively impaired after damage in either of these sites. This discrepancy raises two possibilities which have important implications for the future construction of emotional measures.

We might question the validity of the present emotional measures, though such tests of emotional perception and expression are commonly used in neuropsychological investigations. Certainly, the present tests had high face validity, since they used emotional stimuli, and/or required emotional responses. Furthermore, several deficits in the anterior lesion group could not easily be accounted for in terms of perceptuo-motor or cognitive impairment. Multiple measures also provided validation data. For example, patients with frontal lesions were selectively impaired on all 4 main emotional measures. In addition, qualitative and self-report ratings during card sorting **independently** indicated increased negative reaction in the dysphasic group. Nevertheless, the different emotional measures also occasionally suggested contrary emotional responses, and one interpretation of this is that some (or all) of the multiple measures are invalid. It is therefore concluded that critical appraisal of test validity will be essential in future work. In general, neuropsychological investigators tend to be satisfied with face validity, and to assume that their measures of emotional perception or expression are specifically sensitive to emotional deficits. The use of multiple measures of emotion is uncommon, and cognitive function is not always controlled or subjected to detailed assessment.

The second possibility raised by the absence of deficit on the emotional measures after hypothalamic and medial temporal lesions, is that the present tests may be sensitive to emotional disturbances following frontal lesions, but they may be unable to detect the emotional disorders associated with damage to subcortical sites. Certainly, we know that lesions in different locations can

cause different cognitive deficits (e.g. Jones and Mishkin, 1972; Drewe et al, 1970), and there are indications that distinctive dysphasic syndromes may occur after damage to subcortical structures (Naeser *et al*, 1982; Cappa and Vignolo, 1979). It is quite possible that subcortical lesions similarly cause distinctive emotional deficits. In this case, additional ways of assessing emotional disorder will have to be developed. This conclusion should not be surprising, since limbic lesions have been associated with a wide range of emotional symptoms. It is unlikely that the deficits in emotional perception and expression such as have been measured by the present battery of tests can totally explain the symptoms of depression, euphoria, disinhibition of aggressive or sexual impulses, immoral behaviour and lack of concern for the feelings of others, and so on.

It is of particular interest that most of these latter disturbances are often detected by interviewing the patient's relatives, or are observed in the patient's interpersonal interactions with others. In contrast, the present tests of emotional expression and perception were designed for administration in a clinical interview with the individual patient, where the influence of psychosocial determinants of emotional responses was minimised. With such tight control over psychosocial influences, many emotional symptoms could not be elicited by the present clinical test situation. The crucial importance of social-situational factors is particularly well illustrated in primate studies. The decrease in social interaction after amygdalectomy can only become manifest as the operate interacts with other monkeys. Such disturbed social-emotional behaviour is clearly undetectable when the animal is on its own in a cage. It is therefore suggested that we must seek reliable ways of quantifying the emotional disorders displayed by the patient in his interactions with others. This could be done with greater use of the relative's report of the patient's

behaviour, or patient-spouse interactions may be filmed and analysed. Of course, incorporation of the social dimension will not solve all our problems, and in fact, it will generate new problems. Indeed, it was to avoid these difficulties that the influence of social factors was minimised in the present study. One problem is that the relative's report can be biased and misleading. Furthermore, the emotional disturbance in the patient is not always a simple consequence of the patient's brain lesion. The relative's behaviour can exacerbate the patient's emotional distress (Mulhall, 1978), and a previous study indicated that criticism in the relative was partly responsible for depression in the patient (Weddell, 1986).

Clearly, the neuropsychological investigation of emotional disorder after brain damage is at an early stage. Nevertheless, it seems that we are beginning to develop appropriate methods for their exploration. In the effort to find tests of patients' emotional responses, current measures of emotional perception and expression provide a useful starting point. However, investigation of the emotional responses of individual patients, observed in isolation will have to be complemented by the assessment of emotional symptoms as they occur within a social context.

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This thesis is entirely my own work.

Signed

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## **APPENDIX 1**

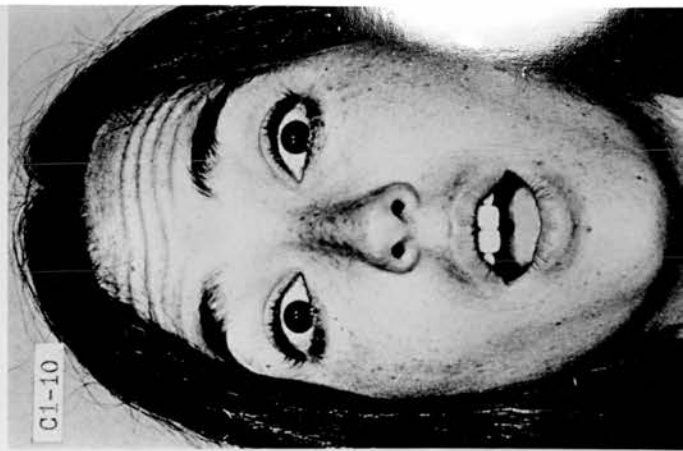
**EXAMPLE ITEMS FROM THE TESTS OF PERCEPTION**

**AND EXPRESSION OF EMOTION**

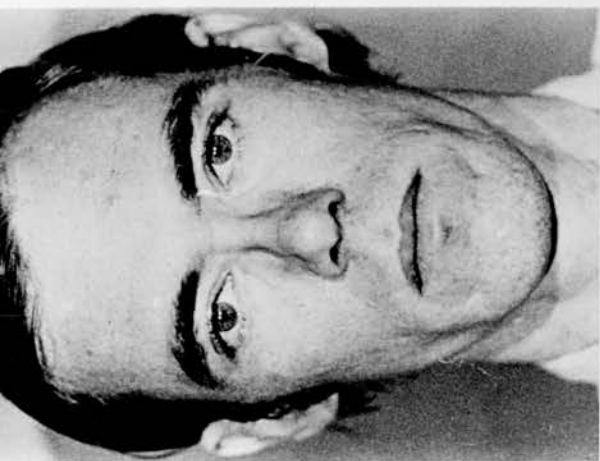
### Test of Perception of Facially Expressed Emotion

Two sets of photographs expressing seven different emotions are given. These were selected from the Ekman and Friesen (1976) series. For all faces used in the test of perception of facially expressed emotion, over 70% of a non-brain damaged sample rated the faces as expressing Sadness, Neutral Feeling, Anger, Disgust, Fear, Happiness, Surprise. The first set of faces are all test items, while the second set of 7 faces were used as the standard response faces in the Face-Face matching conditions of the Test of Perception of Facially Expressed Emotion. (These response faces were also used in the face matching conditions of the Test of Perception of Emotion Expressed in Speech and Text, except that the disgusted and fearful response faces were not used (and were obscured from view) for the latter test.





C1-10



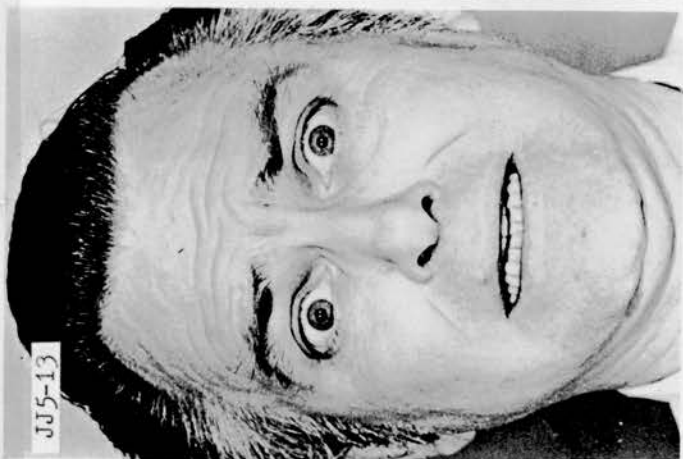
SW3-9



MO1-23



PF1-24



Test of Perception of Emotion Expressed

in Speech and Text: Example test items

Utterances with emotion expressed in tone of voice and in verbal-semantic content:

1. Oh what a perfectly smashing day. (Happy)
2. How lovely Mr Parkinson .... How lovely. (Happy)
3. I can't walk no more ...I can't. (Sad)
4. You shouldn't do this to an old fool like me, you have me in tears. (Sad)
5. Good God, do you never give up. (Angry)
6. Oh stop forcing it on people, Freddie. (Angry)
7. I think they thought they ought to stay. (Neutral)
8. Well goodbye now. (Neutral)

Utterances with emotion expressed only in tone of voice:

1. No, went against the grain. (Happy)
2. Well yes, just a little perhaps. (Happy)
3. Because I want to help you. (Sad)
4. I don't know what it was. (Sad)
5. Roddy was medically unfit. (Angry)
6. I can count up to two. (Angry)

Written sentences with emotion expressed only in verbal-semantic content:

1. What a lovely break. (Happy)
2. You've got a great sense of humour. (Happy)
3. It upsets me to think I'll never see her again. (Sad)
4. That must be awful I really feel sorry for you. (Sad)
5. What on earth do you think you are doing. (Angry)
6. You make me sick. (Angry)
7. How about meeting on Monday. (Neutral)
8. Yes I know. (Neutral)

## The Facial Action Coding System(FACS):

### Examples of FACS Action Units

FACS describes facial expressions in terms of 44 Action Units (AU). AU's can occur in combinations or singly, and each Action Unit is defined in terms of movements of facial landmarks such as the eyebrows and forehead wrinkles (See Figure 1). Since AU's have varying intensities or strengths, the FACS manual describes (a) the extreme manifestations of each AU, as well as (b) precisely specifying the minimum requirement for rating the presence of a particular AU. AU's can be measured reliably, and the FACS manual is organised to provide training in the system of coding. Examples of most of the individual AU's (together with common AU combinations) are provided in photographs or short film clips, to be studied in conjunction with the written descriptions given in the manual. Furthermore, the novice rater can continuously assess the adequacy of his ratings by taking the several tests set throughout the training manual. The present investigator's ratings corresponded reasonably well with the "correct" ratings of the exercise items set in the FACS manual, though it did take 6 weeks of fairly concentrated effort to achieve this standard.

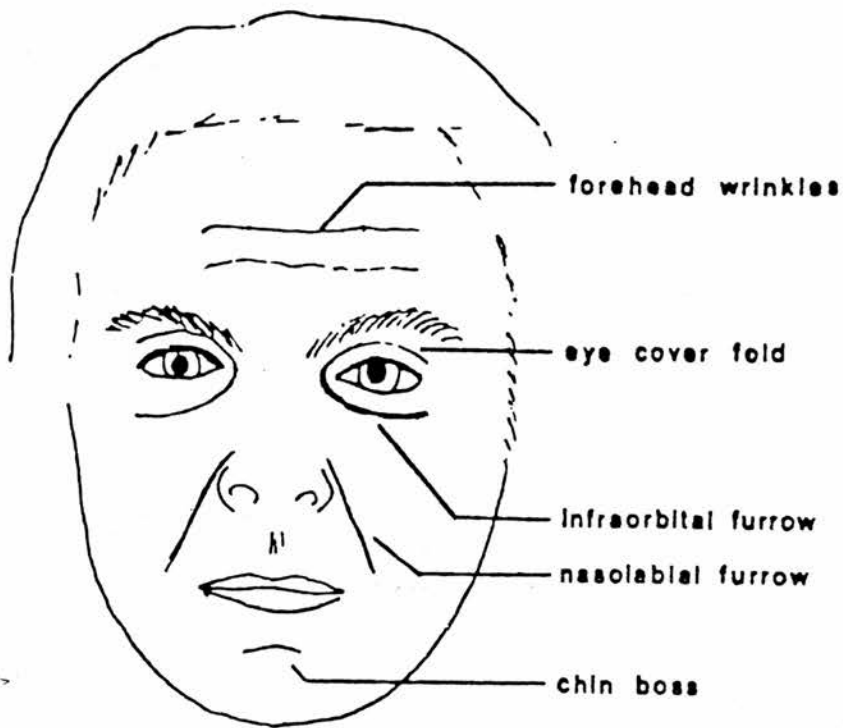


Figure 1

Some of the facial landmarks used in FACS

The minimum requirements for 5 upper and 5 lower face AU, from film, will be given to illustrate the coding system.

#### UPPER FACE AU

##### AU1 - Inner Brow raiser

Inner corners of brows raised slightly; manifest by hair moving or evidence of muscle bulge developing, showing that the inner corner area has been pulled up.

#### AU2 – Outer Brow Raiser

1. Lateral portion of brow pulled upward slightly, changing shape of brow.

and

2. Lateral portion of eye cover fold stretched slightly.

#### AU4 –Brow Lowerer

1. Inner and/or central portion of brow lowered slightly, pushing down or reducing visibility of medial portion of eye cover fold.

or

2. Brows pulled together slightly.

#### AU6 – Cheek Raiser and Lid Compressor

Marked change in either 1 or 2 or slight on both 1 and 2 is sufficient to score 6.

1. Crow's-feet wrinkles; if present in neutral, they must increase.

2. Infraorbital triangle raise: cheeks up, infraorbital furrow deepened, and bags or wrinkles under eyes; if present in neutral, the furrow and either bags or wrinkles under the eyes must increase.

#### AU46 – Wink

1. The eye closure must be unilateral and have a deliberate pause or hesitation.

and

2. The eye closure must be shorter than 2 seconds.

LOWER FACE AUAU9 – Nose Wrinkler

The skin from the medial portion of the infraorbital triangle to the side of the nose is slightly drawn medially and upward towards the bridge of the nose.

AU12 – Lip Corner Puller

1. Skin in the areas of the lower-middle portion of the nasolabial furrow or the furrow itself has been raised up and laterally **slightly**.

and

2. Slight evidence that infraorbital triangle has been raised; most likely will show in lifting and puffing out of lateral top corner of infraorbital triangle.

and

3. Slight evidence that lip corners elongated and angled up. If upward angle is permanent, it must increase slightly.

AU15 – Lip Corner Depressor

1. If the lip line is straight or slightly up in neutral the lip corners moved slightly down.

or

2. If the lip line is slightly or barely down in neutral, then the lip corners moved down slightly more than neutral, and it is not due to AU17 or AU 20.



AU20 – Lip Stretcher

The lip corners and skin adjacent to the lip corners are pulled laterally slightly.

AU28 – Lips Suck

The lips are sucked into the mouth so that the red parts disappear. If this extent of lip disappearance does not occur, consider scoring AU23 or AU32.

## APPENDIX 2

Patient total scores on some measures of emotional perception and expression, together with demographic and lesion data, as well as information on perceptual-cognitive-motor function

Demographic and Lesion Data on Patient Sample

Right anterior							
Case	Age	S	SC	H	Aetiology	Volume	Surgery
1	49	M	3	R	Astrocytoma3/4	97	Lobectomy
2	53	F	2	R	Oligodendroglioma	56	No Surgery
3	35	F	2	R	Oligodendroglioma	39	Lobectomy
Left anterior							
4	64	F	3	R	Astrocytoma3/4	139	Lobectomy
5	45	M	1	R	Astrocytoma3/4	93	Debulk
6	30	M	2	R	Astrocytoma1/2	61	Debulk
7	53	F	4	R	Astrocytoma3/4	110	Debulk
Right posterior							
8	60	F	4	R	Astrocytoma1/2	127	Biopsy
9	64	F	4	R	Unverified	154	No Surgery
10	48	F	3	R	Astrocytoma3/4	97	Biopsy
11	52	M	3	R	Astrocytoma3/4	27	Debulk
12	54	M	4	R	Astrocytoma3/4	154	Lobectomy
13	35	M	3	R	Unverified	134	No Surgery
14	64	F	3	R	Menigioma	117	Excision
15	63	M	3	R	Unverified	100	Biopsy
16	54	M	4	L	Metastasis	32	Excision
Left posterior							
17	58	F	1	L	Menigioma	77	Excision
18	75	M	2	L	Stroke	16	Lobectomy
19	27	F	3	R	AVM	-*	Lobectomy
20	28	M	1	R	AVM	17	Excision
21	63	F	2	R	Stroke	24	No Surgery
22	37	F	2	R	AVM	72	Excision
23	70	M	2	R	Menigioma	139	Excision
24	50	M	2	R	Oligodendroglioma	99	Debulk
Right hemisphere							
25	47	F	3	R	Stroke	61	No Surgery
26	52	M	3	R	Astrocytoma3/4	117	Lobectomy
27	43	M	3	R	Astrocytoma3/4	52	Biopsy
28	25	M	2	R	Astrocytoma1/2	25	Debulk
29	53	F	2	R	Astrocytoma1/2	57	Lobectomy
30	57	M	3	R	Astrocytoma3/4	142	No Surgery
31	58	F	3	R	Astrocytoma3/4	91	Lobectomy

## Left hemisphere

32	48	M	4	L	Unverified	80	No Surgery
33	46	M	3	R	Astrocytoma3/4	89	Lobectomy
34	50	M	3	L	Unverified	109	Biopsy
35	28	M	3	L	Astrocytoma3/4	34	Biopsy
36	61	F	2	R	Meningioma	110	Excision
37	62	F	3	R	Astrocytoma3/4	110	Lobectomy

## Bilateral anterior

38	53	M	3	L	Meningioma	180	Excision
39	48	M	3	R	Unverified	160	Biopsy
40	43	F	2	R	Astrocytoma3/4	75	Lobectomy
41	47	F	3	R	Astrocytoma3/4	98	Debulk
42	61	M	3	R	Astrocytoma3/4	85	Lobectomy
43	63	M	3	R	Astrocytoma3/4	186	Debulk
44	56	F	3	R	Astrocytoma3/4	97	Biopsy
45	47	M	3	R	Astrocytoma3/4	153	Lobectomy

## Bilateral posterior

46	58	M	3	R	Meningioma	100	Excision
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## Basal Forebrain

47	45	F	3	R	Meningioma	-*	Excision
48	44	F	1	R	Craniopharyngioma	-*	Debulk

## Controls

49	25	M	3	L	-	-	-
50	38	M	3	R	-	-	-
51	38	F	3	R	-	-	-
52	44	M	1	R	-	-	-
53	70	M	1	R	-	-	-
54	61	F	3	R	-	-	-
56	62	F	2	L	-	-	-
57	65	F	2	R	-	-	-
58	45	M	3	L	-	-	-
59	40	M	3	L	-	-	-

S - Sex

SC - Social Class

Volume - Lesion Volume (\* - Poor scan,  
lesion volume not estimated)

Performance on Intellectual, Memory, and Perceptuo-Motor Tests

## Right anterior

Case	FIR	FRM	WRM	PIQ	VIQ	PE	SpM	Im	Dys	Neg
1	16	10	19	101	92	33	6	11	-*	0
2	-	22	24	96	98	90	3	5	-	0
3	-	14	4	76	70	85	4	13	-	0

## Left anterior

4	18	11	11	81	65	-	-	-	1	-
5	15	21	14	75	46	78	-	24	1	-
6	-	23	18	101	79	27	0	2	0	-
7	16	24	24	99	69	100	3	16	1	-

## Right posterior

8	-	13	21	91	79	38	2	3	-	0
9	18	11	23	71	80	80	3	9	-	0
10	-	15	12	95	110	-	-	-	-	0
11	-	19	21	76	102	62	2	-	-	0
12	14	13	16	63	106	82	10	0	-	1
13	-	9	22	102	108	0	8	9	-	0
14	18	4	9	91	100	50	2	10	-	0
15	12	13	16	58	77	42	7	7	-	1
16	18	19	22	96	106	31	0	1	-	0

## Left posterior

17	17	20	24	110	86	40	2	3	0	-
18	-	-	-	100	98	0	7	-	0	-
19	16	22	24	89	96	0	11	6	0	-
20	-	15	10	107	93	40	7	1	0	-
21	-	-	-	-	-	-	6	14	1	-
22a**	18	19	6	89	50	-	-	-	1	-
22b**	-	-	-	-	-	70	9	0	0	-
23	18	18	23	101	102	50	3	15	0	-
24	18	24	21	92	80	-	-	-	0	-

## Right hemisphere

25	6	5	12	66	89	33	-	-	-	1
26	14	1	19	70	92	100	-	-	-	-***
27	-	21	21	103	100	83	8	3	-	0
28	18	22	22	122	115	0	0	1	-	0
29	12	12	13	89	102	58	2	10	-	0
30	3	-	-	55	92	58	8	15	-	1
31	6	16	15	62	92	23	0	11	-	1

## Left hemisphere

32	-	13	12	86	64	57	5	8	1	-
33	18	16	2	92	59	-	-	-	1	-
34	18	20	13	92	86	25	3	10	0	-
35	18	21	19	110	93	31	7	1	0	-
36	16	21	14	93	65	93	6	18	1	-
37	-	17	17	84	63	43	8	18	1	-

## Bilateral anterior

38	-	-	-	92	96	29	4	-	-	-
39	13	0	2	69	84	99	4	27	-	-
40	17	17	20	69	98	99	8	1	-	-
41	16	0	2	75	74	91	3	23	-	-
42	18	16	13	93	69	57	0	16	1	-
43	-	13	12	65	92	40	1	-	-	1
44	-	-	-	89	84	99	2	-	-	-
45	-	12	13	96	102	67	2	-	-	-

## Bilateral posterior

46	-	19	15	97	104	30	6	-	-	-
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## Basal forebrain

47	18	15	8	99	98	93	-	10	-	-
48	-	-	-	103	119	36	-	2	-	-

## Controls

49	-	16	23	-	-	50	-	2	-	-
50	-	16	15	-	-	33	-	1	-	-
51	-	-	-	-	-	15	7	-	-	-
52	-	23	24	-	-	50	4	0	-	-
53	18	-	-	-	-	-	-	-	-	-
54	-	24	24	-	-	30	4	1	-	-
55	-	24	24	-	-	50	9	3	-	-
56	-	23	24	-	-	35	12	1	-	-
57	-	24	24	-	-	33	2	4	-	-
58	-	22	22	-	-	33	4	0	-	-

\* - a dash indicates missing data

\*\* - This patient was seen over a 3 week period. She was initially dysphasic, but was not dysphasic during assessment for facial expression. Her score from initial testing and late testing were kept separate.

\*\*\* - This patient had equivocal signs of neglect, and was excluded when comparing neglecting and non-neglecting patients.

FIR - Face Identity Recognition	PE - % Perseverative Errors
FRM - Face Recognition Memory	SpM - Speech Markers
WRM - Word Recognition Memory	Im - Imitation of Facial Movement
PIQ - Performance IQ	Dys - Dyphasia(Present=1,Absent=0)
VIQ - Verbal IQ	Neg - Neglect(Present=1,Absent=0)

Performance on Tests of Emotional Perception and Expression

## Right anterior

Case	FET	VET	SFAC	Wneg	SRneg	HRneg	VFAC
1	16	40	8	6	8	877	14
2	21	50	15	0	2	718	9
3	19	30	9	3	11	1018	13

## Left anterior

4	32	37	-	-	-	-	-
5	14	36	11	11	13	751	7
6	17	43	9	9	5	922	11
7	22	28	6	5	14	592	9

## Right posterior

8	24	42	14	8	7	-	14
9	23	42	23	5	9	792	15
10	12	47	-	-	-	-	-
11	26	39	20	8	4	-	9
12	16	48	20	15	12	808	11
13	22	54	20	1	5	899	14
14	19	40	20	1	5	805	13
15	16	39	31	6	8	822	16
16	21	47	7	2	2	820	11

## Left posterior

17	25	54	15	1	6	741	12
18	-	-	24	15	-	-	-
19	33	63	25	11	9	815	15
20	23	54	19	20	2	-	13
21	30	44	26	23	10	-	16
22a*	25	53	-	-	-	-	-
22b*	-	-	14	5	3	-	18
23	26	47	28	19	9	655	12
24	30	46	-	-	-	-	-

## Right hemisphere

25	10	22	-	-	8	-	-
26	19	27	-	-	10	669	-
27	25	51	11	1	9	-	10
28	37	63	16	0	2	668	10
29	15	29	3	0	4	773	13
30	11	27	4	0	3	586	8
31	12	24	16	4	11	531	5



## Left hemisphere

32	19	48	7	19	13	756	15
33	17	30	-	-	-	-	-
34	26	50	3	3	4	762	14
35	24	51	2	0	8	873	11
36	17	28	13	11	16	877	21
37	12	29	15	13	11	-	15

## Bilateral anterior

38	15	24	10	4	5	-	14
39	7	22	10	4	5	842	3
40	20	47	12	4	9	1255	13
41	2	5	22	13	11	690	8
42	16	25	20	12	8	731	6
43	-	17	0	0	4	-	6
44	11	21	6	5	0	-	9
45	-	36	4	5	3	-	11

## Bilateral posterior

46	24	47	24	14	10	-	14
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## Basal forebrain

47	19	50	24	3	13	594	10
48	28	67	29	-	4	-	-

## Controls

49	29	63	14	10	10	824	9
50	34	60	21	4	15	1057	12
51	29	51	14	4	5	-	12
52	39	65	34	12	1	932	18
53	29	49	-	-	-	-	-
54	36	64	19	5	10	895	11
55	36	58	25	16	8	745	12
56	29	53	26	5	0	860	13
57	35	53	28	8	8	880	14
58	33	60	-	-	8	-	13

\* - This patient was seen over a 3 week period. She was initially dysphasic, but was not dysphasic during assessment for facial expression. Her score from the initial testing and late testing were kept separate.

FET - Perception of Facially Expressed Emotion, Total score

VET - Perception of Verbally Expressed Emotion, Total score

SFAC - Spontaneous FACS, Total AU

VFAC - Voluntary FACS, Total AU

- Wneg - Qualitative Ratings of Negative Reaction after Wrong  
Card Sorts
- SRneg - Composite Self-Report Measure of Negative Reaction  
to Card Sorting
- HRneg - Heart-rate during Wrong Card Sorts